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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

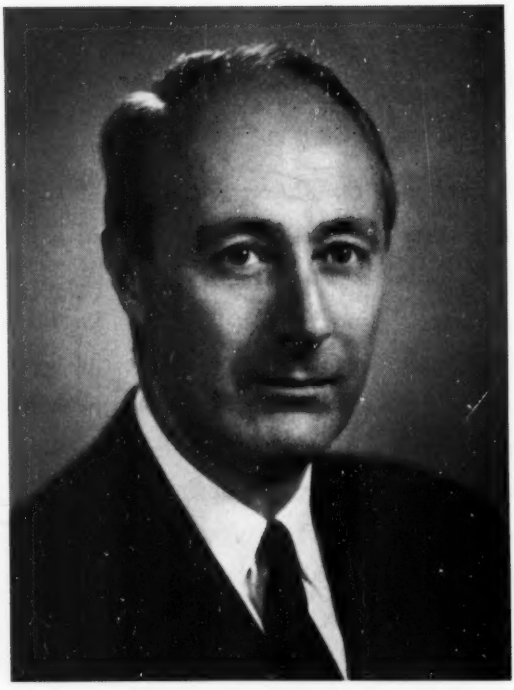
The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

1958 Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

November 6, 7, 8, 1958

Dallas, Texas



SIGURD P. RAMFJORD, D.D.S., M.S., PH.D.

Chairman, Program Committee, American Academy of Periodontology 1958

The program of the meeting of the American Academy of Periodontology which will be held in Dallas November 6, 7 and 8 promises to be outstanding. Many men have worked on this program but much credit goes to the Chairman of the Program Committee, Dr. Sigurd P. Ramfjord.

Dr. Ramfjord was graduated from Oslo University School of Dentistry in 1934, practiced in Oslo 1934 to 1946. He came to the University of Michigan, Ann Arbor, in 1946 and received his M.S. in Periodontics there in 1948 and his Ph.D. in Oral Pathology in 1951. Dr. Ramfjord is a member of a number of Scandinavian and American professional societies and has been active in the American Academy of Periodontology for several years. He is at present Professor of Periodontics, School of Dentistry, University of Michigan.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FIFTY-EIGHT

Oral Medicine
Past, Present and Future*

by LESTER W. BURKET, D.D.S., M.D.,** *Philadelphia, Pennsylvania*

IT is impossible to give a concise and yet inclusive definition of that phase of dental education and dental practice which is commonly known as Oral Medicine or Dental Medicine. This need not cause too much concern since a concise, and yet inclusive definition of the field of internal medicine has not yet been formulated. One can consider, however, those phases of dental practice which require a thorough evaluation and consideration of the health status of the patient in diagnosis general treatment planning and the performing of our professional services to be included in the scope of oral medicine.

In the areas of dental practice included under the heading of oral medicine, the physical well-being of the patient is in most instances the determining factor in treatment planning and in the response to treatment. This field of dental service, as the name suggests, may be closely related to medical problems experienced by our mutual patient. The appreciation, understanding and utilization of the cooperative efforts of the physician and dentist make for a high level of comprehensive health service. It might thus be said that oral medicine represents a bridge between the bio-mechanical phases of dental service and clinical medicine. A bridge, which does not receive the use it should for a better and more comprehensive health service for the patient.

The specific areas of clinical dentistry commonly included within the scope of oral medicine are (a) diagnosis, especially as it pertains to the oral manifestations of systemic disease and the systemic significance of oral disease (b) the field of endodontics (c) the field of periodontics and (d) the ever increasing research activities related to, and associated with these phases of dental practice. The clinical phases of oral medicine, periodontology and endodontics are more closely related to the biologic sciences than to the physical sciences. Hence the patient as a biologic entity becomes the dominant and frequently the limiting factor in treatment and the response to treatment.

What is the present status of the field of oral medicine in dental education and in dental practice? What will be the status of this field ten years from now?

*Presented at the dedication of the dental school of Puerto Rico University, Nov. 14, 1957.

**Dean, School of Dentistry, University of Pennsylvania. Professor of Oral Medicine, School of Dentistry and Graduate School of Medicine, University of Pennsylvania.

It is a most challenging and stimulating assignment to prophesy the place oral medicine might occupy in dentistry a decade hence. Developments in all phases of dental service are taking place with ever increasing rapidity. The refinement of present day technics, the development of new or improved materials or therapeutic agents, the development of new and better instruments will have a marked effect on all phases of dental practice. An additional important consideration is the significant contributions in all areas which can be expected from a greatly expanded research activity. There is no question but what the field of Oral Medicine is moving ahead rapidly, and in my opinion full momentum has not yet been reached. It is likely that our accomplishments and new developments in the field of Oral Medicine in the next decade will be measured more in terms of geometric progression rather than a simple arithmetic progression.

DEVELOPMENT AND PRESENT STATUS OF ORAL MEDICINE

A survey of the development of the field of oral medicine during the past two decades, with which I am personally familiar, may serve as a foundation upon which to make a ten-year projection or prophesy. A brief review of the development of our profession will reveal that in the beginning, the major attention was devoted to the relief of pain, usually by means of removing the aching tooth. Procedures were then developed for filling "holes" in the teeth, or filling the spaces where teeth had been lost through accident or disease. These early restorative services were supplied chiefly for esthetic reasons.

As time passed, more attention was given to the maintenance of health of the oral structures and an increased emphasis given to the restoration of function as well as esthetics in the operative and prosthetic procedures. While there has been much talk about the functional approach in dental treatment, there are still many voids in our knowledge of oral physiology. It is expected that as the science of oral physiology becomes developed that this biologic science will make marked contributions to a better understanding of the causation and treatment of many forms of periodontal disease and other diseases in the field of Oral Medicine.

In the early 1920's, the concept of focal infection captured the imagination of the physician, the dentist and frequently captured, by extraction, the patient's teeth. Many edentulous patients now bear witness to the enthusiasm with which this concept was accepted in clinical medicine. While this intense interest in focal infection unfortunately did not achieve its therapeutic goal, it did call to the attention of the dental profession the potential significance and the many inter-relationships between oral and systemic disease, in addition to the now well developed dental surgical technics and restorative services. The focal infection enthusiasm, thus focused the physicians', the dentists' and the patients' attention on dentistry as an agency of health. In this respect the focal infection era represents an important milestone in the development of dentistry as one of the health services.

ENDODONTICS

The "dead tooth" or the pulpless tooth was considered to be of major clinical importance during the period when focal infection had captured the imagination of the professions. At this time, the treatment of the "dead" or pulpless tooth was not generally accepted by the physician as a desirable health measure nor was this operation always performed by the dentist with a complete understanding or appreciation of the biologic factors involved.

Endodontic treatment advanced markedly during the past two decades with a corresponding improvement in the clinical results. The improved therapeutic results can be ascribed to the more routine employment of microbiologic control procedures, a more complete knowledge of the pathology of the periapical tissues and the

development of new therapeutic agents. Thus, it has become generally conceded even by the sceptical physician as well as the dentist, that a "pulpless" or an abscessed tooth can be treated following accepted surgical technics and bacteriologic control procedures and can be retained without presenting a hazard to the general health of the patient. In fact, in some systemic conditions, endodontic therapy is considered preferable to the extraction of teeth from the standpoint of the patient's general health.

PERIODONTICS

Two decades ago, the treatment of diseases of the gums received little if any emphasis in dental education and in dental practice. The better understanding of the local and systemic factors responsible for periodontal disease, and the associated pathologic changes, have resulted in the development of improved methods of treatment and improved prognosis. While noteworthy achievements have been made in respect to the early recognition of periodontal disease and more effective treatment, our fundamental knowledge in this field of oral medicine is far from satisfactory.

The dental and public health significance of periodontal disease can be better appreciated when it is realized that in the adult more teeth are lost as a result of periodontal disease than as a result of dental caries. The lack of well established preventive measures against periodontal disease, comparable to those currently available for the control of dental caries, emphasizes further the magnitude of the periodontal disease problem. The more widespread employment of caries control procedures will also result in an increasing large number of teeth being retained in late adult life. The tooth population susceptible to periodontal disease will thus be greater in the years to come. The general ageing of the population will also result in more patients surviving into the age range where the morbidity of periodontal disease is highest.

In the phases of Oral Medicine discussed, there is need for increased knowledge about preventive measures and more accurate diagnosis. Laboratory and clinical research is now being conducted in all areas with major emphasis in the field of periodontics.

As a result of the marked accomplishments in the field of oral medicine, along with the accomplishments in the surgical and restorative phases of dental practice, Dentistry today is generally recognized as one of the major health services. The practicing dentist must consider his services primarily in terms of health and not in terms of fillings, bridges or dentures. This broader concept of dentistry as an agency of health, has increased the scope of dentistry and in a corresponding manner the responsibilities of the practicing dentist, particularly in the field of oral medicine which is so closely allied to and concerned with the general health of the patient.

In Dentistry, as in Medicine, increased emphasis is being placed on a positive approach to health and in terms of prevention and the early recognition of disease.

Dentistry's achievements in the field of preventive services are deserving of special comment. For decades, Dentistry has led the way among the health professions in sponsoring and in educating the public about the importance of periodic dental examinations. For many years this has become an accepted phase of dental care. Dentistry can justifiably be proud of this accomplishment.

DIAGNOSIS

In recent years, emphasis has been placed on a more extended oral health service which includes in addition to the examination of the teeth for cavities, a careful evaluation of the supporting tissues for early signs of periodontal disease and a thorough examination of all the oral tissues and exposed areas of the face. The dentist is thus in a strategic position to detect not only diseases of the oral tissues at an early stage but also the oral manifestations which

may be associated with a yet unrecognized systemic disease. The frequency with which diseases of the endocrine system, the blood forming organs and the oral manifestations of nutritional deficiencies are first seen in the mouth emphasizes the general health potentialities of a comprehensive oral examination. Dentists are in a strategic position to detect oral precancerous and cancerous lesions at an early stage.

With the current interest in, and the more general acceptance of the periodic general health examination, it is particularly important that a complete oral health evaluation, made by a qualified individual, be included as a part of the general physical examination. Unless the physician has had special training and experience in stomatology, this part of periodic health service should be performed by a dentist preferably one with special qualifications in the field of oral medicine. It is a great personal concern to observe the frequent omission of a comprehensive oral health evaluation in many of the otherwise excellent periodic general health examinations. It should be stressed that the dentist should be a regular functioning member of the team furnishing health services to the patient.

With the more general appreciation and acceptance in professional circles of Dentistry as a health service, there developed an increased interest in the comprehensive diagnosis of all oral disease including the various lesions of the tongue and the oral mucosal manifestations of local and systemic causation. This more widespread interest in comprehensive diagnosis is a comparatively recent development in dentistry.

In 1925, the first department of oral diagnosis was established in a dental school. Some ten years later, this phase of dental education and practice was still in its infancy. Within the past two decades marked progress has been made in the field of diagnosis. Even now the undergraduate instruction and the students' capabilities in diagnosis and treatment planning manifest striking variations in emphasis and in quality in the different dental schools. The recent dental graduate is generally less confident and less proficient in this important field of health service than in the restorative and prosthetic aspects.

ORAL MEDICINE DURING THE NEXT TEN YEARS

With this historical background of the development of the field of oral medicine, and the comments concerning the present status of this phase of dental practice, let us now consider the possible status and role of oral medicine a decade from today. It is anticipated that dentistry as an agency of health will be universally recognized by both our medical colleagues and our common patients. The dentist will be considered as an indispensable working member of the team responsible for the maintenance of health. The case finding responsibilities of the dentist, and also his potentialities in this respect will be far better developed, more widely appreciated and utilized. The dentist's role in the early recognition of oral changes associated with the important degenerative systemic diseases, nutritional disorders and endocrine disorders will be even more fully appreciated.

By the end of the next decade one can anticipate a considerable change in emphasis of many of the areas included under the heading of oral medicine. There is good reason to believe that there will be a lessened emphasis on endodontics. This will be discussed in greater detail later.

The emphasis on comprehensive diagnostic services and treatment planning will be greater. Patients will accept the dentist as the diagnostician of all oral disease just as patients now accept the physician as a diagnostician of systemic disease. The dentist will gradually become a regular working member of all general health service teams, whether these operate in group medical practice, in diagnostic clinics or in hospitals. It is anticipated that there will be great progress in certain special fields of dental health service concerned with the treatment and rehabilitation of differ-

ent categories of handicapped individuals.

The greatest emphasis during the next decade will be on periodontal disease. This emphasis will be directed toward the development of preventive measures which are applicable on a public health level rather than on an individual basis. As our knowledge of all forms of oral disease becomes more complete, it is anticipated that more effective technics and therapeutic agents will be available to the dentist and his patient.

Research on oral disease in terms of the biologic sciences will move forward rapidly on all fronts. It is desirable that this research activity be both coordinated and long range in nature.

ENDODONTICS IN THE NEXT DECADE

With the more widespread utilization of available methods for caries control it is anticipated that the dental caries work load ten years from now will be less. With fewer carious lesions and increased patient interest in caries preventive measures, the need for endodontic therapy resulting from pulpal infection secondary to dental caries will be markedly reduced. The chief employment of endodontic treatment ten years from now will be for those teeth which have been accidentally injured or for those special instances when the preparation of a tooth for restorative or prosthetic uses will require the removal of the dental pulp. This reduced emphasis on endodontics in the next decade should be taken as a clear cut indication of the development of dentistry as a true health service whose chief interest is in the prevention of disease rather than the treatment of disease.

This suggestion that endodontic therapy will assume a less important role in clinical dentistry during the next decade does not mean that clinical and laboratory studies will not be continued in an attempt to improve further the quality of endodontic services above the present high standards. The current standards of endodontic treatment are now generally recognized by the physician as not jeopardizing the general health of the patient. In the next decade, there may be more systemic conditions in which endodontic therapy is considered preferable from a general health standpoint to the extraction of the teeth.

PERIODONTICS IN THE NEXT DECADE

There is every indication that before the end of the next decade the prophylactic measures against periodontal lesions and the treatment of this important group of diseases will occupy a place of major importance in dental practice. Methods of evaluating the extent and degree of periodontal involvement have only recently been developed and employed for epidemiologic purposes. This has been a long needed development for determining not only the magnitude of the periodontal problem but more important for serving as a basis for evaluating the effectiveness of newly developed preventive measures or therapeutic procedures.

It is anticipated that a decade from today the treatment of periodontal disease will become a major activity of the general practitioner of dentistry. It will also be one of the truly challenging problems of dental practice because of the time consuming nature of periodontal therapy and the fact that auxiliary personnel can do little to ease the work load in treatment. Moreover the final outcome of periodontal treatment is not controlled to the same degree by the skill of the dentist as the usual restorative or prosthetic services. The results of periodontal therapy are also more dependent upon the physiologic response of the patient—a highly variable factor and one which in our present state of knowledge is extremely difficult to predict. The ageing of our population with its increased tooth population will also increase the need for periodontal treatment.

While specialists in the treatment of periodontal disease, the periodontists, will still have an important place in dental practice, it is believed that the major work load of periodontal treatment must be and

will be assumed by the general practitioner of dentistry. Periodontal treatment will become one of the regular phases of general practice, with only the more difficult patient, or those with the most advanced periodontal disease being referred to the periodontist. This awakened interest in and increased attention to the treatment of periodontal disease are now already developing.

The current increased emphasis placed on periodontal disease in dental education will also have a marked effect on the practice of this phase of dentistry within the next decade. The innumerable important inter-relationships between restorative, orthodontic and prosthetic dental services in the causation and treatment of periodontal disease will become more generally appreciated by the dental practitioner and the specialist. With the development of this appreciation, there is every reason to believe that the major objective of all forms of restorative dental services will be directed towards the prevention of periodontal disease in the later years of life.

Some examples can be cited to show how this more general interest in the prevention of periodontal disease may develop. It is anticipated that within the next decade even greater emphasis will be placed upon the functional relations of the restorative and prosthetic services rendered by the dentist. This will require markedly expanded research on the physiologic relations of the jaws during rest and function. The science of oral physiology will become developed as one of the most important special fields in the biologic sciences.

The pedodontist or one treating dental disease in children will not only direct his attention to the immediate problems at hand, but in addition will consider these in terms of the prevention of periodontal disease later in life. The orthodontist will not only strive for a good esthetic result following treatment, but he will adjust the occlusion to achieve an harmonious functional relationship at the conclusion of treatment. These examples are cited to indicate the great interdependence and interrelationship of all phases of dental practice in the prevention and treatment of periodontal disease.

COMPREHENSIVE ORAL EXAMINATIONS AND CASE FINDING RESPONSIBILITIES IN THE NEXT DECADE

For decades, Dentistry has lead the way among the health professions in sponsoring and educating the public to the importance of periodic dental examinations. During the next decade more emphasis will be placed on a comprehensive oral health service which includes a thorough examination of all the oral structures and exposed areas of the face. The dentists' strategic position in detecting early signs of oral disease and the oral manifestations which may be associated with a yet undiagnosed systemic condition will become more generally recognized. The frequency with which diseases of the endocrine system, the blood forming organs and the oral manifestations of nutritional disease are first seen in the mouth will be recognized as evidence of the general health significance of a comprehensive oral examination made by the dentist.

It is anticipated that in the next ten years the significance and importance of comprehensive diagnosis and treatment planning will be universally appreciated by the dentist and recognized by the patient. The potentialities of the dentist, well oriented in the field of oral medicine, as a case finder for systemic disease will not only be appreciated but utilized widely. Dentists will become a part of the working team conducting general diagnostic clinics. It is not unlikely that members of the dental profession with advanced training and experience in the diagnostic phases of this profession will be included as a unit of group-practice teams in medicine. Unless the physician has had special training and experience in stomatology, this phase of periodic health service should be performed by a dentist, preferably one with special qualifications in oral pathology.

DENTISTRY IN HOSPITAL PRACTICE IN THE NEXT DECADE

During the next decade the dentist will become a regular and indispensable member of the team furnishing health services in hospitals. Today there is a common tendency to consider oral surgery in hospital practice as being synonymous with Dentistry. The accomplishments of oral surgery as a specialty of Dentistry, especially in hospital practice, are generally recognized. It should be understood however that this represents only one facet of dentistry. There is a rapidly developing appreciation that for the hospitalized patient Dentistry has a much broader scope and responsibility as an agency of health. It is anticipated that the broader scope of dental services in hospital practice will be recognized more generally by the development of Departments of Dentistry. These Departments of Dentistry will furnish all the different dental services available in dental office practice and in addition those specialized services required by the hospitalized patients and those dental services which can be most effectively performed only in the hospital.

While the dentist is more likely to observe oral lesions in private practice which are suggestive of as yet unrecognized systemic disease, it is not unusual for the dental member of the hospital team to note changes in the soft oral tissues or in the dental or jaw roentgenograms which furnish a clue to the systemic complaints of the patient. The case finding potentialities of the dentist in hospital practice will become more generally accepted.

Dentistry's role in hospital practice during the next ten years will have a vastly expanded scope and a correspondingly increased responsibility. The preoperative preparation of the oral cavity of all general surgical patients will become a routine procedure. The hospitalization of certain groups of patients, such as the incorrigible dental patient, the spastic or cerebral palsy patient and other handicapped patients, for restorative dental services will be employed more generally.

As the population ages and greater interest and emphasis are placed upon geriatrics in medical practice and hospital care, Dentistry will be called upon to play an important role in the comprehensive treatment of these patients. It is recognized that many of the problems of the ageing population are those related to an inadequate masticatory system, functionally inadequate artificial dentures or in many instances the lack of artificial dentures.

Other phases of oral medicine will become more generally recognized and developed in the next decade. In this discussion, the area of oral medicine is assumed to include those dental services which are required for the general physical and social rehabilitation of the patient. The dentist will be generally accepted as the "watch dog" of the oral cavity, its contained and associated structures as far as the recognition of precancerous and cancerous lesions are concerned. While he may not be qualified to treat these lesions unless he has had special training in surgery or radiation, he will be generally recognized as an indispensable member of the team in planning treatment of oral malignancies, and in assisting in the treatment of these lesions.

Dentistry has an important role in the rehabilitation of other groups of patients. The dentist's role in the social rehabilitation of the patient who has had extensive surgical procedures involving the orofacial area for the eradication of cancer will have become generally recognized. Dentistry's role has already been clearly established in the treatment and rehabilitation of the cleft palate patient. It is anticipated as a result of current research that even greater achievements will be made in the years ahead in this area.

The dentist will assume a more important role in the next decade in all problems associated with speech defects. It should be emphasized that the structures

associated with speech are in the field of responsibility of the dentist. Dentists are now developing speech aids which far excel those previously available. These are superior in terms of intelligibility and social acceptance, and more important they require only a fraction of the time formerly needed for learning to use the speech aid.

While these dental services are not commonly considered under the field of oral medicine, they do represent areas where the closest cooperation between the dentist and the physician are necessary for the most effective treatment of the patient.

SUMMARY

The predictions made by those appearing on this program can be evaluated only in terms of developments in the future. Most certainly all of our predictions will not become actualities. It is realistic however, to expect that the next decade is one of great promise in Dentistry.

The increased emphasis on basic and applied research, further refinements of new instruments for performing tooth cutting operations and the development of new dental materials will result in notable progress during the next ten years.

With an ever increasing demand for dental services by a public that is enjoying an unequaled standard of living, one might assume that the road ahead for Dentistry will be both wide and smooth. Dentistry will no doubt enjoy increased professional stature and public respect during the next ten years, but the road ahead will undoubtedly have some rough spots and an occasional detour.

The members of our profession should constantly remember that they are given by law an exclusive franchise, to practice dentistry. This franchise in the form of licensure is given on the assumption that this is necessary to assure the public that a high standard of health service will be furnished. The dental profession must be always mindful that the service they render to the public is their sole justification for existence as a privileged professional group. While the acquisition of new knowledge and the development of new and improved methods for treatment of dental caries and periodontal disease are laudable accomplishments, they are not by themselves justification for the continued existence of Dentistry as a health profession.

It is important that during the years ahead we not only maintain but enhance the respect of the public for our profession. It is also important that every practicing dentist become a public relations representative for the dental profession, and not a personal promotions agent for himself.

If at any time a large segment of the public should consider that the interests of the dental profession are predominantly selfish in nature, the privileged status now enjoyed by the dentist could be changed by legislation. It is unfortunate that the profession as a group is too often judged by the ill-advised actions of a few. It is important that the ethical conduct of our profession advances in harmony with our research and technological developments.

Another "rough spot" in the highway of our professional development is rapidly approaching. With the more widespread use of auxiliary personnel, who have a higher level of educational background, there exists the possibility that the more aggressive auxiliary personnel may attempt to assume some of the privileges now enjoyed by the formally educated dentist. Legal attempts will no doubt be made by certain groups of auxiliary personnel to obtain the right to perform some of the procedures which are now the responsibility and legal prerogatives of the dentist.

The chief arguments of these individuals is that they can perform the same service to the public at a lower fee. Activities such as these could result in the development of two recognized levels and possibly standards of dental service. At best it could cause the patient, who is the public, to wonder whether the duly licensed dentist is actually worthy of the fees asked.

This problem of auxiliary personnel possibly assuming some of the prerogatives of the dentist is unlikely in the field of oral medicine. This field, where the evaluation of the health status of the patient plays such an important role, is not one which can be readily taken over by auxiliary personnel.

Potential problems may exist however in the field of oral medicine between the dentist and the physician. This is understandable since the boundary between the fields of oral medicine and internal medicine is not well defined nor clearly understood. It is conceivable that an opportunist in the field of oral medicine might attempt to establish too extensive boundaries. Physicians who are unaware of the scope of the present day education of the dentist in oral medicine might misinterpret the case finding activities of his dental colleagues.

It is essential that both the physician and the dentist have a definite understanding relative to the limitations and abilities of both professions. Only through mutual respect and cooperation on the part of both the physician and the dentist can the highest level of health service be made available to the patient.

The important role the research worker will play in the development of our profession in the next decade has been discussed. It has been mentioned and stressed that every practicing dentist must be a public relations representative for dentistry. Through service of a high standard and exemplary conduct, our profession must continue to maintain the respect of the public we serve.

There is another important group in our profession upon whom the future development of our profession is also dependent; namely, the dental teacher. Our profession owes a great debt of gratitude to the teachers in Dentistry. The development of dentistry in the years ahead will depend in a large measure on their unselfish interest and indefatigable efforts at a great personal and family sacrifice. Today, and for many years, the hard working teacher has been subsidizing education to a greater extent than all the philanthropic foundations combined.

It is my sincere hope that within the next decade the dental teachers will achieve the recognition and respect of the dental profession that they deserve. It is also hoped that before the next ten years have elapsed that the faculty members in our dental schools will receive a compensation commensurate with their contributions to the profession and that they will enjoy a justly deserved economic security.

AMERICAN ACADEMY OF DENTAL MEDICINE

The American Academy of Dental Medicine will hold its Thirteenth Mid-Annual Meeting and Luncheon at the Hotel New Yorker, New York City, on Sunday, December 7th, 1958. There will be a business meeting for members at 10:00 A.M. The afternoon scientific session will be devoted to "The Gingival Tissue." Participating in this program will be: Henry M. Goldman, D.M.D., Director of the Riesman Dental Clinic, Beth Israel Hospital, Boston; Professor of Periodontology and Chairman of the Department of Periodontology, Graduate School of Medicine, University of Pennsylvania. Maury Massler, D.D.S., Professor and Chairman of the Department of Pedodontics, University of Illinois. Stanley D. Tylman, D.D.S., Professor and Chairman of the Crown and Bridge Department, University of Illinois.

All members and interested dentists and physicians are cordially invited to attend. For reservations and copies of program, please address the Academy Secretary, Dr. Walter I. Levine, 809 Cathedral Street, Baltimore, Md.

Cartilage Grafts in Human Periodontal Pockets

by ERWIN M. SCHAFFER, D.D.S., M.S.D., *Minneapolis, Minn.*

IN an attempt to restore alveolar bone, investigations were made of cartilage grafts in monkeys.[1] In some instances, the cartilage was grafted fresh, and others were implanted after the cartilage had been preserved in alcohol. From this investigation, it was found that fresh autografts and homeografts of cartilage placed in surgical periodontal pockets were non-viable. Of course, the fixed cartilage was non-vital. Although the cartilage did not grow, both the fresh and fixed grafts acted as a template around which new bone formed. The cartilage was invaded by connective tissue. The new bone that formed was seen on the surface of the cartilage and in the surrounding connective tissues. It was evident that the bone extended by apposition into the cartilage after the invasion by connective tissue. The new bone, which extended from the surface into the center of the cartilage, could be seen filling the space between and within the lacunae which was formerly occupied by chondrocytes. There were no major differences between the autografts and the homeografts.

From this study, it was felt that cartilage grafts in human periodontal pockets should be investigated. Some questions were raised in the monkey investigation, namely: Can new bone be formed around and within cartilage grafts in human periodontal pockets; will the new attachment following grafting be coronal to the old attachment; will any given individual accept a homeograft of cartilage in the periodontium; is there any difference in the rate of wound healing? The following clinical investigation was undertaken to answer these questions.

Clinical Professor, Division of Periodontics, University of Minnesota School of Dentistry, Minneapolis, Minnesota.

PROCEDURES

In 26 patients, 70 homeografts of cartilage were done. The grafts were done after the cartilage had been preserved in 70% ethyl alcohol, and stored in a refrigerator for a varying number of days, from 7 to 90. The source of the cartilage was from the noses of other patients following submucus resections. The patients and the pockets were selected at random from navy and marine personnel at the Marine Corp Base, Camp Pendleton, California. The ages of the patients varied from 18 to 56. All of the patients were in good health, with one exception. This exception was a marine private who was receiving treatment for multiple boils (#14 M 10). The pockets used in this survey were selected at random and varied in depth from 3 to 12 millimeters. Forty-one were of the supracrestal type and 29 were subcrestal pockets. One of the pockets was complicated by bifurcation (#19 M 5) and another by trifurcation (#12 D 14) involvement.

Color photographs and preoperative x-rays with a periodontal probe extending to the bottom of the pocket were taken. Prior to treatment, the following measurements were recorded: (1) the depth of the pocket and, (2) the distance from the gingival margin to a reference point either the cervical line or the tip of the cusp. After infiltration with a local anesthetic, a new attachment operation was done.[2] This operation consisted of: (1) a partial gingivectomy to remove the marginal epithelium; (2) root curettage to remove all deposits, and to establish a smooth, hard surface; (3) curettage to remove all of the soft tissue in the subcrestal defect including the epithelium and connective tissues. However, in supracrestal pockets, the curettage was more limited than in sub-

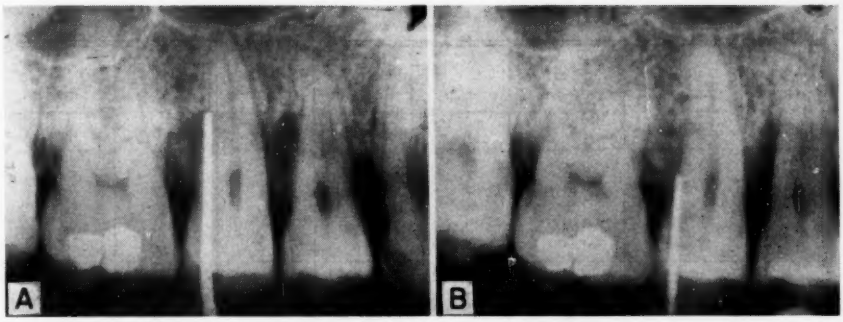


Fig. 1. A. Preoperative (#15 D 13)—8/8/55. B. Postoperative (#15 D 13)—7/7/56.

crestal pockets, but it included the curettement of the epithelial lining and attachments; (4) final root planing using curettes in a vertical, horizontal and diagonal direction with emphasis on push strokes; (5) inserting cartilage to fill the space created by the soft tissue curettage. Prior to grafting, the cartilage was removed from the alcohol, and any remnants of connective tissue were removed by scraping with a sterile scalpel. The cartilage was then placed in normal saline for 1 hour; (6) protecting the wound, after a blood clot had formed around the cartilage, with a periodontal pack to which terramycin was added. Care was taken to make sure that the pack was not placed between the wound and the root. After placing the pack, it was lifted, which permitted one to peek and to insure that the pack was on the surface of the wound and *not in* it. The pack was removed 72 hours later to make sure that a blood clot or granulations covered the cartilage graft. In the few instances in which the cartilage could be seen, bleeding was produced by puncturing the surrounding tissue. Thus, a new blood clot was formed on the surface of the cartilage. The blood clot and the wound was protected in all cases for a minimum of two weeks. Any given pack was not permitted to remain over seven days. All of the grafts were done, with the above direct technique of insertion, with the exception of 13. In these 13, a flap was laid after the partial gingivectomy. Then, all of the other steps followed in proper sequence. Finally, after the cartilage was implanted,

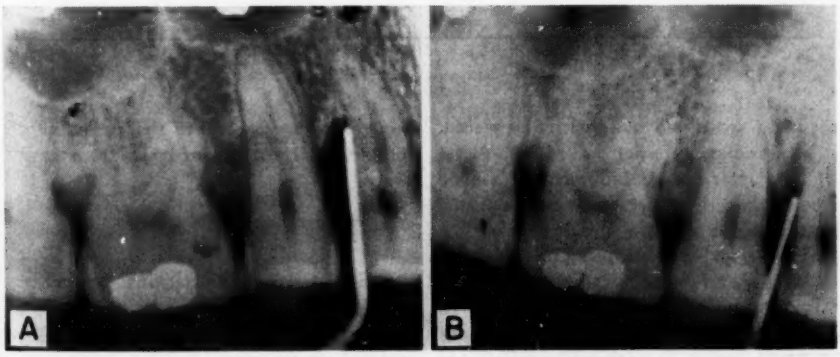


Fig. 2. A. Preoperative (#15 D 12)—8/8/55. B. Postoperative (#15 D 12)—7/7/56.

TABLE I

Patient #	Pocket Location	Before	After	Recession	New Attachment	Type of Pocket	P.O. X-Ray* Findings
#1	M Li 4	8	2	1	5	Sub	NB
#2	M 7	5	2	0	3	Sup	NC
	D 8	5	3	0	2	Sup	NC
	D 9	5	2	0	3	Sup	NC
	M 10	5	3	0	2	Sup	NC
	M 14	7	3	0	4	Sup	NB
#3	Li 6	9	2	3	4	Sub	No X
	D 6	7	3	2	2	Sub	No X
	La 9	4	2	1	1	Sup	No X
	La 8	5	1.5	2.75	2.75	Sup	No X
	M 8	5	2.5	0	2.5	Sup	No X
#4	D 5	5	3	2	0	Sup	CC
#5	La 7	6	5	1	0	Sup	No X
#6	D 28	10	5	4	1	Sub	NC
#7	M Bu 18	7	3	1	3	Sub	No X
	M 30	7.5	5	3	−0.5	Sub	No X
#8	D 7	5	3	0	2	Sup	NC
	Li 9	5	2	0	3	Sup	ND
	Li 10	5	1	0	4	Sup	ND
	M Li 31	6	3	1	2	Sup	NC
#9	M 10	11	3	2.5	5.5	Sub	NB
	M 7	7.5	2.5	1	4.5	Sup	NB
	M 24	7	3	1.5	2.5	Sup	NB
	M 25	7	3	1.5	2.5	Sup	NB
	M 3	7	4	0	3	Sub	NB
	M 14	6	4	0.5	1.5	Sub	NB
#10	Li 11	5	2.5	0	2.5	Sup	No X
#11	D Bu 19	7	3	1	3	Sub	NB
	M 24	5	3	0	2	Sub	CC
#12	D 11	7	3	2	2	Sup	NB
	D 5	6	3	3	0	Sub	NC
	D 14	5	3	0	2	Sup	NC
#13	D 14	7	2.5	1.5	3	Sup	No X
	M 15	7	2.5	1.5	3	Sup	No X

the flap was sutured and a pack was placed.

The wounds were not probed for at least 21 days after the establishment of a good blood clot. Postoperative measurements of the sulcus and the distance from the gingival margin to the same reference point were recorded. Kodachromes were taken postoperatively in all cases, and postoperative x-rays were possible in 58 of the 70 grafts. The other 12 grafts could not be x-rayed because the patients had been transferred or separated from active duty.

RESULTS

The cartilage was tolerated by the recipient tissues and there was no apparent foreign body reaction. However, in 8 of the 70 grafts, the cartilage degenerated.

TABLE I

Patient #	*Pocket Location*	*Before*	*After*	*Recession*	*New Attachment*	*Type of Pocket*	*P.O. X-Ray* Findings*
#14	M 10	6	6	0	0	Sub	No X
#15	D 6	12	7	4	1	Sub	NC
	La 6	5	2	1	2	Sup	ND
	D 5	5	2	1	2	Sup	ND
	D 4	5	4	0.5	0.5	Sup	CC
	D 12	7	3	1	3	Sub	NB
	D 13	5	3	0.5	1.5	Sub	NB
#16	M Li 30	5	2	2	1	Sup	NC
	M Bu 30	7	2	1	4	Sup	NC
	Li 4	7	2	1	4	Sub	ND
	M Li 4	8	5	1	2	Sub	NB
	M Bu	8	5	2	1	Sub	NB
#17	Li 21	6	2.5	2	1.5	Sup	ND
	M Li 25	7	3	2	2	Sup	CC
#18	Li 12	5	2	0.5	2.5	Sub	NB
#19	M Li 21	5	2	0.5	2.5	Sup	NC
	D Li 21	7	2	2	3	Sup	NC
	Li 5	7.5	5	0	2.5	Sup	ND
	M 5	7	6	1	0	Sup	NC
	D 5	7	4	1	·2	Sup	NB
#20	La 11	5	1	1	3	Sup	ND
#21	Li 29	5	1	1.5	2.5	Sup	NC
	Li 21	3	1	1	1	Sub	NB
#22	M Bu 4	5	3	1	1	Sub	NC
	M Li 4	7	3	4	0	Sub	NC
	D 9	8.5	4	1	3.5	Sub	NB
	Li 9	5	2	2.5	0.5	Sub	NB
	D 7	5	2	2	1	Sup	CC
	M 22	6	5	1.5	−0.5	Sub	NC
#23	D 9	5	2.5	1.5	1	Sup	NC
	M 10	5	2	1	2	Sup	CC
	M 27	3	2	1	0	Sup	NC
	D 26	3	2	1	0	Sup	NC
#24	M 8	5	1	1	3	Sub	CC
#25	M 25	9	2	1	6	Sub	NB
#26	M 6	6	3	1	2	Sub	NB

*NB—New bone coronal to preoperative x-ray
NC—No change
No X—No postoperative x-ray
CC—New cortical crest

Sup—Supracrestal
Sub—Subcrestal
ND—Not diagnostic

(#3 D 6, #5 La 7, #14 M 10, #18 Li 12, #19 D 5, #22 D 9, #22 Li 9, #22 M 22). The degeneration of 5 can be explained as follows: One patient had recurrent necrotizing ulcerative gingivitis (#14 M 10), in one the flap procedure was wrong and it became necrotic (#3 D 6), in two an acute inflammatory reaction followed improper curettage (#22 D 9) (#22 Li 9), in one an acute inflammation

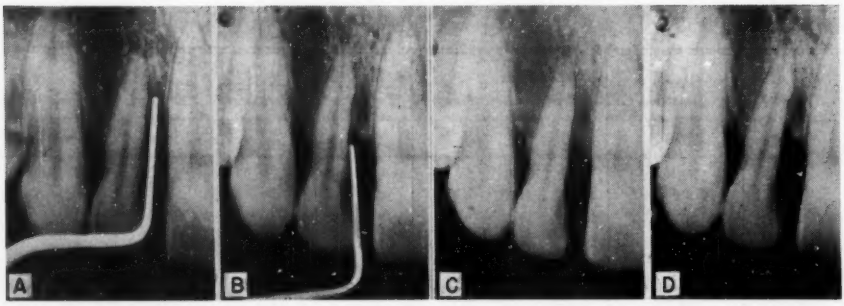


Fig. 3. A. Preoperative (#9 M 10)—5/2/55. B. Postoperative (#9 M 10)—7/2/56. C. Preoperative (#9 M 10)—5/2/55. D. Postoperative (#9 M 10)—7/2/56.

followed the infiltration of a local anesthetic with a contaminated needle (#22 M 22).

Although it could not be positively determined, the degeneration in the other 3 cases was probably the fault of the operator. This seems evident since 2 of these pockets were recuretted and new grafts were inserted which were successful (#18 Li 12, #19 D 5).

The degeneration was not a foreign body reaction because 3 of 6 grafts were successful in patient #22 and in addition, regrafts in two pockets after a second curettage healed uneventfully (#18 Li 12, #19 D 5).

The cartilage appeared to degenerate when there was an absence of either a blood clot or granulation tissue on the cartilage nearest the surface of the wound. Whenever there was an absence of a blood clot or granulation tissue the wound was recuretted, this was necessary in 12 cases, (#6 D 28, #7 MBu 18, #7 M 30, #11 D Bu 19, #15 D 6, #15 D 12, #12 D 14, #16 Li 4, #16 M & Li 4, #16 M Bu, #22 M Bu 4, #22 M Li 4). With the exception of two (#7 M 30, #22 M Li 4) of the 12 cases, a new attachment was evident by probing.

The new attachments varied from 0.5 mm to 6 mm (Table I). However, in 8, there was no new attachment. In 2 the attachment was more apical by 0.5 mm than before treatment (#7 M 30, #22 M 22) but the pocket depth after treatment was decreased, 2.5 mm and 1 mm respectively, by recession. The recession following the treatments varied from 0 mm to 4 mm (Table I) and the sulci depth after treatment varied from 1 mm to 7 mm (Table I).

In 22 the postoperative x-ray did not reveal any change in the bone, i.e., the density was the same and the height of the alveolar bone was neither decreased nor increased. In 7 a new cortical crest of bone was observed postoperatively in which it had been absent prior to treatment. In 21 new bone could be seen on the postoperative x-ray. In these cases the new bone was coronal to the bone before treatment (Figs. 1, 2, 3). In 8, it could not be determined whether or not there was any change in the bone because of the limitations of the x-ray techniques. In these instances the areas treated were on the lingual or buccal surfaces of the teeth.

DISCUSSION

In this study, it seemed evident that the host tissue will accept and tolerate homogenous cartilage grafts. This was apparent from the lack of any foreign body reaction. Although 8 of the 70 grafts degenerated, it was due to the shortcomings of the operator. These failings were improper curettage, flap procedures and grafting before necrotizing ulcerative lesions had

completely healed. If these 8 cases were to be done again, degeneration may be prevented by a more careful selection of cases for treatment and by a more careful operative technique.

Prior to grafting, the wound must be very carefully prepared using a new attachment procedure. This includes the removal of the marginal epithelium; complete and thorough root curettage to remove all deposits and to obtain a smooth, hard surface; thorough tissue curettage to remove the epithelial lining and attachment; and the protection of the blood clot and granulation tissues during healing. One advantage of using cartilage over bone grafts is that the cartilage can be shaped to fit the wound area. The operator must be very careful not to use too much cartilage. If this is done, it is difficult to get the blood clot to cover the coronal portion of the graft. For a graft to be successful, the granulation tissue must cover the portion of the cartilage closest to the wound surface. It appears to be advantageous to remove the pack periodically during the first week to make certain that a good thick pad of blood clot covers the cartilage. If the clot does not cover the cartilage, the adjacent tissues should be pierced to produce bleeding and the desired clot. At the end of two weeks of healing, if the granulations do not appear to proximate the root surface, it should be recuretted. These procedures were done in this study and it is my opinion that if we hadn't taken care to create and protect an adequate blood clot, our percentage of success would have been greatly reduced.

One possible advantage of using cartilage grafts in the new attachment operation is that the total amount of blood clot necessary to fill the wound is greatly reduced; hence, there is less chance for the blood clot to shrink. Thus, it aids in sealing the wound. The problem of getting granulation tissue to fill the wound appears to be reduced.

Perhaps the most difficult phase of this study was the problem of obtaining postoperative radiographs identical in angle and position and density as compared with the preoperative radiographs. Whenever there was any doubt about the formation of new bone, namely the increase in height of the alveolar process, the postoperative x-ray results were recorded as no change. The preoperative as compared with the postoperative x-rays were evaluated by another periodontist. It is unfortunate that we do not have a completely standardized technique so one could evaluate scientifically the formation of new bone as measured by the radiographs.

Some clinicians and researchers are of the opinion that it is dangerous to remove the epithelium in periodontal pockets because it may result in an increase in pocket depth. This was not evident in this study because in only two cases the bottom of the pocket moved apically from its former site. The new attachment in most cases completely offset this.

This study was designed to determine the effect of cartilage grafts in the new attachment operation. It is not a controlled study. In other words, this type of treatment with grafts was not compared with the new attachment operation without grafts. Consequently, it is impossible to express any opinion on whether the grafting is an aid or a hindrance in obtaining a new attachment. Ideally, it would be well to compare grafting in the new attachment operation with the new attachment operation without grafts.

SUMMARY

In 26 patients, 70 homografts of cartilage were done. The graft was done in the wound after the new attachment operation in periodontal pockets. Prior to grafting, the cartilage was removed from the 70% ethyl alcohol and placed in normal saline for one hour. The ages of the hosts varied from 18 to 56. The pockets treated included 41 supracrestal and 29 subcrestal.

The results of the grafts were determined by measuring the pockets before and after treatment and carefully recording the amount of recession that occurred. X-rays were used to determine the effect of the grafts on the alveolar process. Fifty-eight postoperative x-rays were taken. A new cortical crest was observed in 7 and new bone coronal to the old was evident in 21. In 22, no change could be determined radiographically because of the limitations of the x-ray technique. Some new attachment coronal to the preexisting attachment was obtained in 60 of the 70 grafts. The new attachment varied from 0.5 mm. to 6 mm. Recession following the treatment occurred in 55 of the 70 cases, and it varied from 0.5 mm. to 4 mm. The pockets before grafting varied in depth from 3 mm. to 12 mm. The sulci after treatment varied in depth from 1 mm. to 7 mm. After grafting, pockets with depths of 4 mm. to 7 mm. still existed in 15 of the cases. However, the sulci measured 3 mm. or less in 55 of the 70 pockets.

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Histochemical Changes in Inflammatory Periodontal Disease*

by S. SIGMUND STAHL, D.D.S., M.S.,** HENRY C. SANDLER, D.M.D., M.P.H.,*** AND E. SUBEN, A.B.****

THE histologic changes occurring in periodontal disease have been described primarily as processes of chronic inflammation, degeneration and repair of local tissues.[1,2,3,4] This investigation was concerned with some of the histochemical changes that occur in chronic inflammatory periodontal disease.

MATERIALS AND METHODS

Seven patients, ages 52 to 61, with confirmed diagnoses of squamous cell carcinoma affecting the tongue were treated by surgical excision. The surgical procedure required hemimandiblectomy, but in no case was the mandible or overlying mucosa involved with neoplastic disease. A thorough clinical and radiographic examination of the oral structures included evaluation of periodontal status. The teeth involved consisted of five anterior teeth, eight cuspids, eleven bicuspids and six molars. A diagnosis of periodontitis was established in all instances.

Due to the lack of human mandibles showing no evidence of periodontal disease, normal control sections could not be studied. The observations presented in this investigation are thus based on serial sections which were subjected to various histologic and histochemical procedures for an evaluation and comparison of the resultant staining reactions. The bodies of the mandibles with the teeth in situ were divided into convenient blocks, containing one or two teeth each, immediately after surgery. The blocks were fixed in formalin and subsequently decalcified by the formic acid-sodium citrate method. They were embedded in celloidin, sectioned at 12 microns and stained with hematoxylin-eosin as a base stain.

Fig. 1. Photomicrograph of an interdental papilla of a mesiodistal section of the first and second molars. Mgn. 50x. Hematoxylin-eosin stain. Note the proliferation of the epithelial attachment and round cell accumulation below the area of attachment.

The tissues were also subjected to a number of other histochemical stains. Serial sections of the various blocks were utilized for comparative histological evaluations.

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**Attending Periodontist, VA Hospital, Brooklyn, N. Y.

***Chief, Dental Service, VA Hospital, Brooklyn, N. Y.

****Histology Technician, VA Hospital, Brooklyn, N. Y.

Fig. 2. Photomicrograph of a mesiodistal section of a mandibular second molar. Mgn. 30x. Hematoxylin-eosin stain. Note compression of the periodontal membrane below A and at B.

HISTOLOGIC OBSERVATIONS

Hemotoxylin-eosin stain: Gingival inflammation with ulceration, apical proliferation of the epithelial attachment and resorption of the interdental alveolar crest were common findings (Fig. 1). In many areas examined, crushing of the periodontal membrane, probably as a result of occlusal trauma, also was observed (Fig. 2). In some areas of ulceration the gingival collagen fibers appeared to be degenerating (Fig. 3). It was of incidental interest to note that in almost all of the sections, the insertion of the transeptal fibers was at a level just below the most apical position of the epithelial attachment (Fig. 4) and these appeared to be less affected.

Stains visualizing carbohydrate-protein complexes: A marked positive periodic acid-Schiff reaction was seen in the fibers nearest the area of inflammation, which became less intense as the level of the alveolar crest was reached (Fig. 5). This fuchsinophilia was also observed in similar areas when the allochrome stain was used, except that with this latter staining procedure, a reddish aggregate was observed surrounding the bluish staining fibers (Figs. 6, 7, 8). There were fewer fiber bundles visible at the site of ulceration than near the alveolar crest level (Figs. 9, 10). When stained by silver impregnation, there was a marked increase in argyrophilic fibers near the area of ulceration as compared with the more apical areas (Figs. 11, 12, 13). Metachromatic staining with toluidine blue showed the area of ulceration to be either orthochromatic or mildly beta metachromatic. Enzyme depolymerization with testicular hyaluronidase in experimental and serial control sections was introduced for various periods of time and followed by PAS stain. It was noted that in the experimental sections the fuchsinophilic aggregate became

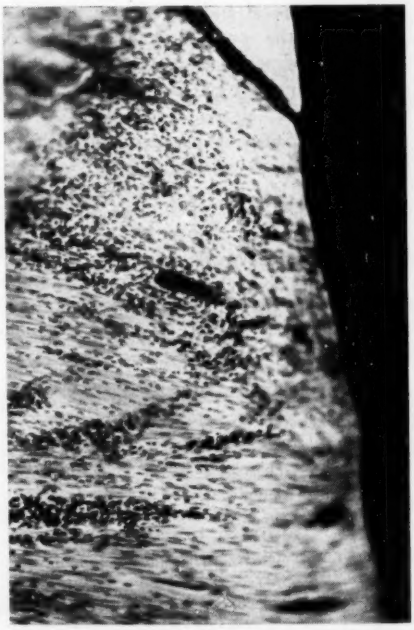

Fig. 3. Photomicrograph of area of epithelial attachment in a mesiodistal section of a mandibular first molar. Mgn. 100x. Hematoxylin-eosin stain. Note the round cell infiltration below the epithelial attachment and disorganization of collagen fibers.

somewhat less PAS positive after three hours of depolymerization. Depolymerization for 24 hours changed the appearance to PAS negative, but some fibers still stained fuchsinophilic (Fig. 14).

The areas of crushing of the periodontal membrane also showed a PAS positive reaction in the membrane (Fig. 15) which stained orthochromatically with toluidine blue and showed little hyaluronic acid lability. PAS positive staining reactions were further observed in the connective tissue surrounding areas of bone resorption, but were less intense in areas of osteogenesis (Fig. 16).

Fig. 4. Photomicrograph of area of epithelial attachment in a mesiodistal section of a mandibular first molar. Mgn. 100x. Van Giesson stain. Note the attachment of the transeptal fibers at a level just below the most apical end of the epithelial attachment, (E.A.).

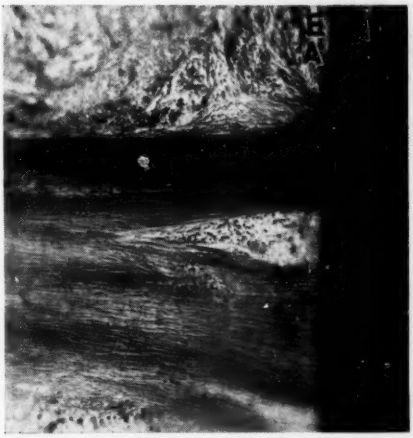

Fig. 5. Photomicrograph of the same area of transeptal fiber attachment in a mesiodistal section of a mandibular first molar shown in Fig. 4. Mgn. 75x (serial section). PAS stain. Note the marked dark appearing PAS positive reaction in the fiber bundles near the apical level of the epithelial attachment, (E.A.).

DISCUSSION

Theoretical considerations: The periodic acid-Schiff reaction has generally been used to visualize carbohydrate-protein complexes in ground substance.[5,6,7] Lipids may also yield a positive PAS reaction. However, since this reaction is relatively unexplored, our discussion will deal primarily with the carbohydrate-protein complexes. The ground substance in connective tissues is said to consist primarily of chondroitin sulfuric acids, hyaluronic acid and mucoproteins.[8] In normal tissues, the ground substance is not easily stainable with the periodic acid-Schiff reaction nor is it metachromatic with toluidine blue.[9] It has been noted in studies of normal human gingiva that collagen bundles of the corium tend to be metachromatic, but that the degree of metachromasia varies considerably.[10,11]

Studies of inflamed gingiva have been reported to show a diminution of PAS positive ground substance in chronic gingivitis with a tendency toward disintegration of the basement membrane,[12] although the latter may also disappear under normal conditions.[13] Marked PAS positive and

Fig. 6. Photomicrograph of the same area of transeptal fiber attachment in a mesiodistal section of a mandibular first molar shown in Fig. 4. Mgn. 75x, (serial section). Allochrome stain. Note the marked dark appearing PAS positive reaction in the fiber bundles apical to the epithelial attachment, (E.A.).

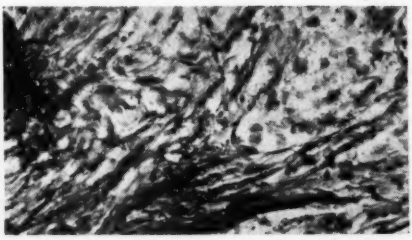

Fig. 7. Photomicrograph of areas of dark staining fibers shown in Fig. 6. Mgn. 400x. Allochrome stain. Note the diffuse PAS positive staining material around the fibers.

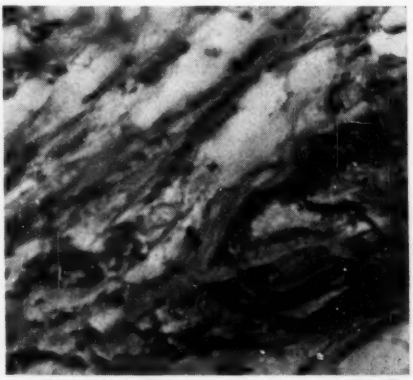

Fig. 8. Photomicrograph of higher magnification of fibers shown in Fig. 7. Mgn. 990x. Allochrome stain. Note the crumbling of fibers and the diffuse PAS positive material seen in the area surrounding the fibers.

metachromatic material however, have consistently been observed in pathologic skin lesions such as in granulation tissue, skin wounds, and stroma of tumors.[14,15,16,17,18,31] Some investigators have described PAS positive and metachromatic material as localized in the ground substance between fibers[20,21] while others are of the opinion that this material constituted a coating of the fibers themselves.[22,23] Electron microscopic studies provide additional support to the concept that the ground substance coats the fibers under some pathological conditions.[24,25] In the specimens described in this study, aggregates of PAS positive material between the fibers as well as fuchsinophilic fibers could be observed throughout the area of inflammation. When the fuchsinophilic fibers were subjected to silver impregnation, they were usually argyrophilic, i.e., they are interpreted as reticulum fibers.[26,27,28,29,30] This fuchsinophilia and metachromasia of the fibers is less evident with progressive collagen bundle formation.[31]

Experimental Observations: In the specimens under study, a marked increase of argyrophilic fibers was noted near the area of ulceration, which decreased as the level of the alveolar bone crest was approached.

Fig. 9. Photomicrograph of transeptal fibers below the epithelial attachment, (E.A.). Mgn. 200x. Allochrome stain. Note the relative reduction in collagen bundles.

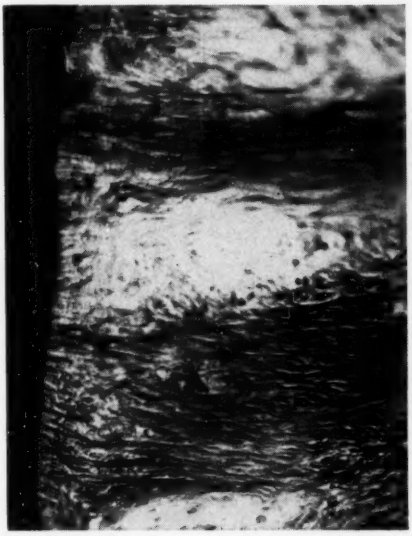

Fig. 10. Photomicrograph of transeptal fibers apically to those shown in Fig. 9, and nearer the alveolar crest. Mgn. 200x. Allochrome stain. Note the relative increase in collagen bundles in the area.

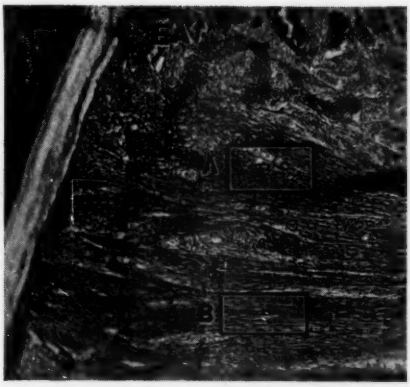

Fig. 11. Photomicrograph of transeptal fibers shown in Fig. 5. Mgn. 75x. Wilder silver impregnation.

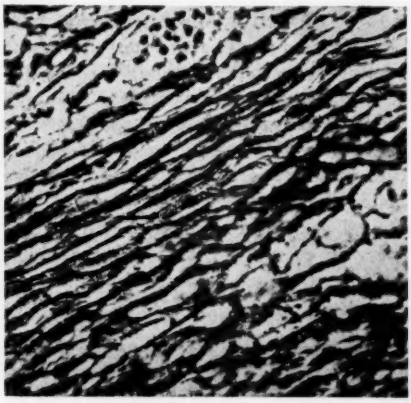

Fig. 12. Photomicrograph of area A in Fig. 11. Mgn. 250x. Wilder silver impregnation. Note the increased argyrophilic fibers.

Destruction of fibers as well as repair as demonstrated by an increase of young (i.e. reticulum) fibers, seem to take place simultaneously in periodontal inflammation.

Further attempts at identification of the visualized fuchsinophilic material, showed it to be slightly beta metachromatic and partially hyaluronidase labile. Among PAS positive material, free acid mucopolysaccharides are known to give a gamma red metachromatic reaction. Hyaluronic acid does not produce tissue metachromasia.[32, 33,34] Enzymatic digestion with hyaluronidase followed by staining with the periodic acid-Schiff procedure, while not specific, will often show a reduced fuchsinophilia of hyaluronidase labile material.[35] It would thus seem that the fuchsinophilic material visualized in our study may be composed of mucoproteins and hyaluronic acid. Increased hyaluronidase content with diffuse

Fig. 13. Photomicrograph of area B in Fig. 11. Mgn. 250x. Wilder silver impregnation. Note reduction in argyrophilic fibers as compared with Fig. 12.

PAS positive staining material has been demonstrated in inflammation and edema of the skin which was thought to be due to mucins having been diffused from the inflammatory reticulum or the basement membrane.[19, 36, 37] Gersh and Catchpole[38] believe that this increase in PAS positive material in areas of inflammation and around growing tumors is due to the appearance of water soluble glycoproteins, resulting from the depolymerization of the ground substance by enzymes such as hyaluronidase and mucinases. It must be pointed out however, that there is still no agreement regarding this depolymerization process, since as Meyer[39] emphasizes, the increased fuchsinophilic material could also be the result of a new substance or an increase of a substance present prior to inflammation.

The changes taking place in the ground substance around growing tumors and at sites of inflammation among other lesions, have been thought to facilitate the spread of the lesion.[40, 41] Increases in the fuchsinophilia of the ground substance has also been observed in the tissue surrounding erupting teeth, as well as in desquamative gingivitis.[42, 43] It thus might be postulated that the increased lability of the ground substance observed in areas of periodontal inflammation may well influence the speed of apical proliferation of the epithelial attachment. In this connection, the recent investigations focusing attention on the role of bacterially produced proteolytic enzymes which appear to be present in greater amounts in areas of gingival inflammation[44, 45, 46, 47] are of particular interest to the study of tissue changes in periodontal inflammation.

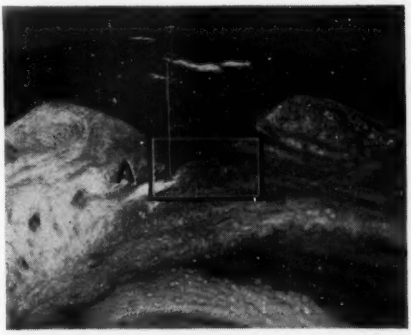

Fig. 15. Photomicrograph of interradicular septum of a mandibular molar. Mgn. 100x. Allochrome stain. Note the dark appearing PAS positive area at A where the periodontal membrane is narrowed.

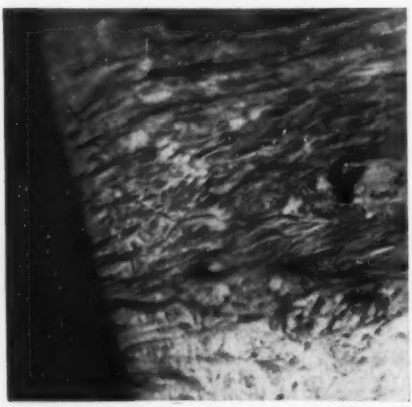

Fig. 14. Photomicrograph of area of transeptal fiber attachment. Mgn. 250x. 24 hour hyaluronidase depolymerization followed by PAS stain. Note the presence of dark appearing PAS positive fibers.

The observation of fuchsinophilic mate-

rial in areas of periodontal membrane crushing due to occlusal stresses, may be an indication of the activity of enzymes originating from the involved tissue cells and acting on the ground substance.[48]

The visualization of PAS positive material in the ground substance surrounding bone activity in our material, has also been noted in experimental animal studies[49, 50, 51] and points to a relation between mucopolysaccharide lability and alterations of bone.

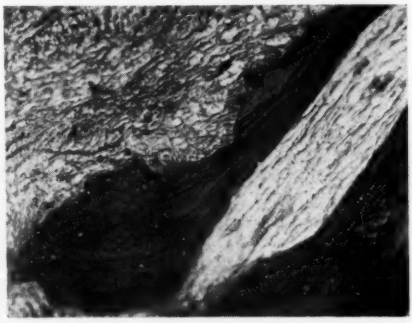

Fig. 16. Photomicrograph of an area of alveolar bone and periodontal membrane in a mesiodistal section of a lower molar. Mgn. 200x. Allochrome stain. Note the diffuse dark appearing PAS positive material in the area of osteoclastic resorption.

A comment should be made regarding the validity of histochemical observations in decalcified materials. It is obvious that long term decalcification of large segments of calcified structure will destroy some of the tissue components, such as glycogen in short order. A recent study comparing the effects of various decalcifying agents on the histochemical behavior of bone and cartilage has shown that PAS positive substances undergo little modification, except for glycogen after exposure to acid decalcification.[52]

It is thus felt that while a reduction of fuchsinophilic tissue components takes place during section preparation, PAS positive material is retained and may be observed following the decalcifying procedures.

SUMMARY AND CONCLUSION

Serial sections of thirty human teeth and their supporting structures were subjected to various histochemical analyses.

1. It was noted that in most areas of gingival inflammation, marked fuchsinophilic aggregates could be observed following staining with the periodic acid-Schiff and allochrome procedures. These fuchsinophilic aggregates stained orthochromatically and sometimes beta-metachromatically, and were partially hyaluranidase labile. An increase of argyrophilic fibers was also observed in the area of inflammation.

2. In many areas of narrowing and crushing of the periodontal membrane, probably due to excessive occlusal stresses, fuchsinophilic material could be visualized.

3. Areas of alveolar bone resorption were characterized by the presence of fuchsinophilic staining material in the ground substance. A lessened fuchsinophilia of the ground substance was observed in areas of osteogenesis.

The response to injury and repair found in chronic inflammatory periodontal disease seemed to be characterized histochemically by an increase in the visualization of mucopolysaccharides and an associated increase in reticulum fibers. The visualized mucopolysaccharides are likely to be composed of mucoproteins and hyaluranic acid.

The authors wish to thank Dr. Karl Steiner and Dr. Lester R. Cahn for their advice in the preparation of this study, and Mr. Richard Gilliam for the preparation of the photomicrographs.

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A Clinical Evaluation of a Desensitizing Toothpaste

by PATRICK D. TOTO, D.D.S., M.S., HARRY STAFFILENO, D.D.S., ANTHONY W. GARGIULO, D.D.S.,* *Chicago, Illinois*

A commercially available dentifrice useful in controlling the complaint of sensitive dentine by home brushing is a convenient and frequently desirable product. Such a product, Thermodent,** was secured and tested under controlled conditions for its dentine sensitivity prevention and control properties.

One area of dental therapeutics frequently attended by the complaints of sensitive dentine is in periodontal therapy. The process of thorough subgingival and supragingival planing of the root surfaces cuts away nonvital cementum and the underlying dentine. This frequently results in some teeth which are sensitive to air temperature changes in the oral cavity, to the introduction of acid foods, the increase in osmotic pressure resulting from increase in organic acid and other carbohydrate degradation products on the sensitive root surface and to simple physical irritation by exploration or brushing. It was decided to test the effectiveness of Thermodent upon those patients about to receive periodontal therapy.

MATERIAL AND METHOD

Seventy-one adults including forty-one women and thirty men selected for treatment of chronic periodontitis were examined. A record of their oral hygiene, brushing habits, technic and sensitive teeth was secured.

The oral hygiene determined by observing the accumulations of materia alba and stain on the teeth was reported as poor, good, or excellent. Brushing habits were determined by the number of times each day that the teeth were brushed; the length of time in minutes; whether before or after meals; and a determination of acceptability of the brushing technic. In this case the brushing technic was recorded as acceptable or non-acceptable. Also, the use of tooth picks, dental tape and the type of dentifrice was recorded.

Sensitive teeth were recorded as to their number and location; onset preoperative or postoperative; and termination. The location of sensitive teeth was divided into six regions. They are the upper right molar and premolar, lower right molar and premolar, upper left molar and premolar, lower left molar and premolar, the upper six anteriors, and the lower six anteriors. Each region was scored only once when sensitive teeth were found.

To determine the effectiveness of Thermodent as a preventive and a control of hypersensitive dentine, the subjects were divided into two groups: one group of forty-seven subjects were given Thermodent while twenty-four were given a placebo. The subjects were instructed to brush their teeth after each meal for three minutes. Neither the subjects nor the examiners were aware of the character of the dentifrice. A code assigned to each dentifrice tube was known only to one of us (P.D.T.). The placebo consisted of the same ingredients as Thermodent except for the omission of 1.4% formalin. The subjects used their dentifrice for a period of five weeks during which they underwent periodontal therapy. All of the subjects were treated by root planing. In addition, twenty-three subjects underwent gingivectomy. Basically, the toothpaste consisted of:

Thermodent:	*Placebo:*
PPT Chalk	PPT Chalk
Formalin 1.4%	Formalin 0.0%
Salts	Salts
Flavoring	Flavoring

*Foundation for Dental Research, Loyola University Dental School.

**The material used for this study was supplied by the Thos. Leeming Co.

Frequency Distribution

No.	*Av. Age*	*Oral Hygiene Brushing Habits*								
		P.	*G.*	*E.*	*T/D*	*Av. Length*	*AC*	*PC*	*A*	*NA*
41 Women	40	22	17	2	2	2 min.	18	22	12	28
30 Men	40	13	14	3	2	2 min.	7	21	15	13

P—Poor
G—Good
E—Excellent
T/D—Times each day
AC—Before meals
PC—After meals
A—Acceptable
NA—Non-acceptable

Figure 1

FINDINGS

There were forty-one women and thirty men; the age ranges were between twenty-five years and sixty-six years. The average age of the group was about forty years; the oral hygiene ranged from poor to good while five subjects scored excellent. Twenty-two women or thirty-eight per cent and thirteen men or eighteen per cent showed poor oral hygiene while seven per cent showed excellent oral hygiene. Both the men and women averaged two tooth brushings each day with a brushing time of two minutes. Twenty-five subjects brushed their teeth before meals, forty-three after meals and three did not brush their teeth at all. Forty-one subjects, fifty-seven per cent, had non-acceptable brushing habits while twenty-seven, thirty-eight per cent, showed acceptable habits. The ratio of poor oral hygiene to non-acceptable brushing technique was thirty-three to forty-one.

The complaint of sensitivity was observed preoperatively in forty-six subjects or sixty-four per cent. Postoperative sensitivity was found in fourteen subjects or nineteen per cent. Eleven subjects or seventeen per cent never experienced the complaint of sensitivity.

The forty-six subjects who complained of sensitive teeth preoperatively showed a definite aggravation of the complaint after the first week of periodontal treatment. Thus, sensitive teeth became hypersensitive. There was a total of two hundred and sixty-eight sensitive teeth which averaged 5.8 teeth per subject. Such sensitive teeth were regionally distributed as follows: $\frac{11\ \ 9\ \ 13}{11\ \ 9\ \ 12}$. The fourteen postoperative sensitive subjects showed a total of sixty-seven hypersensitive teeth which averaged four teeth per subject. The regional distribution of such teeth was as follows: $\frac{7\ \ 4\ \ 4}{10\ \ 8\ \ 8}$.

The therapeutic response showed a close correlation to the dentifrice used. Thirty-

Sensitivity Frequency Distribution

	No.	*%*	*Number Sensitive Teeth*	*Av. No. Sen. teeth*	*Regions*
Preoperative	46	64	268	5.8	$\frac{11\ \ 9\ \ 13}{11\ \ 9\ \ 12}$
Postoperative	14	19	67	4.0	$\frac{7\ \ 4\ \ 4}{10\ \ 8\ \ 8}$
No Sensitivity	11	17	0	0	

Figure 2

two of the preoperative sensitive group were given Thermodent, while fourteen were given the placebo. The complaint of hypersensitive dentine was brought under control after the second to third week of therapy with an average time of two and one-half weeks, in the experimental group. While there was a diminution of sensitivity after using the placebo, the subjects were not free from the complaint after five weeks of therapy.

Therapeutic Response

		Number	*RP*	*RPG*	*Onset Weeks*	*Term. Weeks*
Preoperative	T	32	21	11	0	2.5
	P	14	11	3	0	0
Postoperative	T	9	8	1	2	3*
	P	5	2	3	1	0
No Sensitivity	T	6	3	3		0
	P	5	3	2		0

*One of the subjects failed to secure complete freedom from sensitivity after five weeks.
RP Root Planing
RPG Root Planing & Gingivectomy
T Thermodent
P Placebo

Figure 3

Nine of the postoperative sensitive group were given Thermodent and five were given the placebo. The onset of the complaint of hypersensitive teeth ranged from the first to the third week of therapy with an average of two weeks in the Thermodent group. The complaint terminated between the second and fifth weeks with an average of three weeks of therapy. The onset of the complaint of hypersensitive dentine was one week in placebo subjects. While there was diminution of sensitivity the group was not free from the complaint after five weeks of therapy.

Six of the subjects who never experienced the complaint of hypersensitive teeth were given Thermodent while five were given the placebo.

DISCUSSION

Our data showed that the patients requiring periodontal treatment averaged forty years of age. All of these selected patients had exhibited a condition of chronic periodontitis. The incidence of periodontal disease was closely correlated to the presence of poor oral hygiene. It was found that forty-six per cent of the subjects presented poor oral hygiene and non-acceptable brushing habits. This evidence supports the continued need for the dentist to teach his patients proper brushing techniques and to encourage good oral hygiene.

It was observed that patients with periodontal disease showed an incidence of sensitive teeth mainly before periodontal therapy. Such sensitive teeth were aggravated by scaling. The frequency ratio of subjects with sensitive teeth was forty-six preoperative to fourteen postoperative. It can be recommended that a careful history and examination of sensitive teeth be done prior to the commencement of periodontal therapy. It was found that sensitive teeth may be located in all regions of the mouth; and that on an average the sensitive subjects had five such teeth. The posterior regions were found to be more frequently sensitive than the anteriors. Following periodontal therapy, it appeared that the lower regions show a greater frequency of sensitivity than the upper regions.

Forty of forty-one patients with sensitive teeth were free of the complaint after three weeks of Thermodent therapy. However, while nineteen subjects with sensitive teeth experienced diminution of sensitivity, they were not free from the complaint after five weeks on a placebo. It is recommended that a desensitizing dentifrice be used as an adjunct during periodontal therapy in order to bring under control the complaint of sensitivity.

Magnetic Instrument for the Recovery of Broken Curette Fragments and Other Steel Foreign Bodies

by MURRAY SCHWARTZ, A.B., D.D.S.,* *New York, N. Y.*

EVEN the most careful operator occasionally breaks an instrument while operating. During the curettage of deep periodontal pockets curette tips may fracture. This may be associated with (a) faulty instrument design or construction or (b) thinning of the blade through repeated sharpening. The methods employed to remove a fragment include flushing the pocket with a jet of water, curetting, and packing gauze into the pocket and pulling it out in an attempt to trap the tip in the gauze mesh. The instrument described below is offered as an additional aid.

Magnets of various designs have been used for centuries in surgery for the removal of iron and steel foreign bodies. When a fragment is embedded in tissue, such as in the eye, a very great magnetic force is needed to remove it. If, however, the fragment is loosely held, as in a periodontal pocket, a properly designed magnet of only moderate strength is needed.

The shaft of this instrument consists of a brass sleeve (A) (4½" long, $\frac{9}{32}$" inside diameter) which holds two permanent magnets (B) made of Alnico V. Each magnet is a rod, ¼" in diameter and approximately ¾" in length. The working ends (C) are steel and are held to the magnets by magnetic attraction. The magnetic force is transmitted through the steel. The steel shanks are held firmly in the brass sleeve by acrylic resin (D). A wooden spacer (E) keeps the magnets firmly in contact with their steel shanks and prevents their breaking away if the instrument should be dropped.

The steel ends may be made in any desired shape from a worn-out or broken instrument. Two limitations must be kept in mind. The end must be made as broad and blunt as possible, and the shank should have as few sharp angles as possible in order to obtain the maximum magnetic force at the tip.

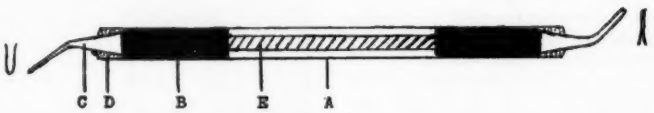

Fig. 1 -(A) Brass sleeve. (B) Alnico magnet, (C) Steel shank. (D) Acrylic resin. (E) Wooden spacer.

The instrument may be sterilized by immersion in cold sterilizing solution. It should not be heated, since heat will soften the acrylic and may weaken the magnets.

This magnetic instrument is used by inserting the end into the pocket in the area where the fragment is located. It is then moved from side to side like a probe, withdrawn, and examined. If the steel fragment has been encountered or closely approached, and it is not embedded in tissue, the magnet will generally remove it.

*Division of Periodontology, School of Dental and Oral Surgery of the Faculty of Medicine, Columbia University, New York, N. Y.

Early Carcinoma of the Gingiva Simulating Gingival Hyperplasia

HAROLD R. SCHREIBER, A.B., D.D.S.,* *St. Louis, Missouri*
CHARLES A. WALDRON, D.D.S., M.S.D.,** *Atlanta, Georgia*

EPIDERMOID carcinoma is the most common type of malignancy of the oral cavity. It has been shown that about 50% of patients with oral carcinoma will first consult a dentist with complaints relative to the presence of the lesion. More important from a control standpoint, the alert oral diagnostician is often in a position to detect these lesions in their early, asymptomatic stages.

The case to be presented is considered unusual because of the age and sex of the patient and the clinical features of an early epidermoid carcinoma of the gingiva.

CASE REPORT

The patient, a white female, 28 years of age, was seen in March, 1957. The medical history was entirely negative. Her dental history revealed that she had been examined by her dentist in July, 1955. At that time the gingival tissues were normal, and the alveolar crests of bone about all the teeth were intact as observed in radiographs made then. Teeth with carious lesions were restored in August and September, 1955. The patient did not see her dentist again until October, 1956, when she was experiencing discomfort in the lower right third molar tooth. At that visit she told her dentist she had noticed a redness and an occasional bleeding of the gingival tissues about the upper anterior teeth for about two months. The patient was referred to an exodontist for extraction of the upper and lower right third molar teeth. She was told then that the redness of the gingival tissues about the anterior teeth probably was due to trauma from an edge-to-edge bite of the anterior teeth.

The patient, although referred by her dentist for further examination in October, 1956, did not make an appointment until four months later. Examination then revealed that the oral mucous membranes were essentially normal with the exception of the labial gingival tissues about the upper right central incisor, upper left central and lateral incisor teeth. Here the gingiva were moderately inflamed, hyperplastic and had lost all normal form and texture. The gingiva appeared to hang as a thin curtain of soft red tissue over the labial surfaces of the three incisor teeth (Fig. 1). One could easily insert a periodontal probe under the labial tissue for about 6 mm. and lift the curtain of tissue. The lingual tissue appeared normal in color, texture, and form, and could not be probed. The anterior teeth were in an edge-to-edge bite but were not in traumatic relationship. A radiograph of the upper anterior area at this time exhibited an unusual amount of alveolar bone resorption about the three incisor teeth, in contrast to radiographs taken only 20 months previously (Fig. 2). Only slight mobility of these teeth was present. Palpation for lymph nodes revealed a small firm node lying at the base of the mandible in the lower left first molar area. The patient claimed that she had been aware of this node for three or four months.

At the following appointment a biopsy of the gingival tissue on the labial surface of the upper right central incisor, upper left central and lateral incisor teeth was performed. A surgical pack was placed over the excised area.

Fig. 1. Clinical photograph taken prior to the excision biopsy showing enlargement of the gingival tissues above the upper anterior teeth.

The specimen was submitted for microscopic examination. The gross specimen consisted of a strip of gingival tissue 5 x 3 mm. in size. There was nothing distinctive on gross examination. The microscopic features, however, were quite unusual. Part of the specimen showed the typical features of chronic gingivitis. The covering epithelium was hyperplastic and the underlying lamina propria showed a heavy infiltration of lymphocytes and plasma cells. At one margin of the specimen there was a very abrupt change in the character of the epithelium. Marked dyskeratosis was present. This was characterized by large, hyperchromatic, atypical epithelial cells, some of which contained abnormal mitotic figures. The depth of excision was such that the extent of invasion could not be

*Washington University School of Dentistry.
**Emory University School of Dentistry.

Fig. 2. Left—Radiograph taken of upper anterior region in July, 1955.

Fig. 2. Right—Radiograph taken at the time of biopsy 20 months after the first film showing the extent of bone loss about these teeth.

determined but the biopsy showed unequivocal evidence of malignancy (Fig. 3 and 4). A diagnosis of epidermoid carcinoma of the gingiva was made. The patient was referred to a surgeon who biopsied the firm submaxillary lymph node. A frozen section showed that this node contained a large focus of metastatic epidermoid carcinoma. A block excision of the premaxilla and a left radical neck dissection were performed. Microscopic examination showed that none of the additional lymph nodes contained metastatic carcinoma. The maxillectomy specimen showed an epidermoid carcinoma arising from the gingival epithelium with invasion of the underlying bone (Fig. 5). The medial and superior margins of the bone excision appeared adequate but islands of tumor cells appeared very close to the lateral line of excision. An additional tooth and block of alveolar process was excised. Microscopic examination of the tissue failed to disclose evidence of carcinoma. Shortly after release from the hospital the patient was fitted with a prosthesis to replace the missing teeth and alveolar process. Recovery to date has been uneventful.

DISCUSSION

Study of the biopsy specimen and numerous sections from the surgical specimen indicate that this tumor arose from a small area of attached and free gingiva. Although the area of the surface involvement was

Fig. 3. Low power photomicrograph of original biopsy specimen showing a strip of relatively normal gingival epithelium on the superior aspect with invasion of the carcinoma into the underlying lamina propria.

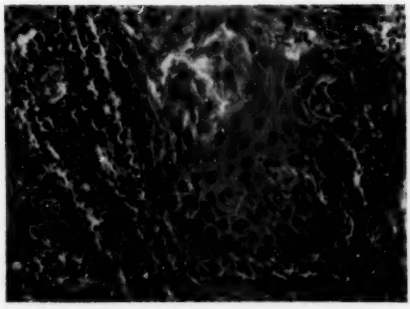

Fig. 4. Medium power photomicrograph of original biopsy specimen showing a mass of carcinoma cells.

Fig. 5. Low power photomicrograph of a section of gingiva from the resection specimen. Normal surface epithelium is present at the lateral margins and the deeply invading carcinoma appears to be arising from a relatively small surface field.

less than 1.0 cm. in diameter, extensive bone invasion was present and the tumor had metastasized to a regional lymph node. It is far too early to speculate as to the ultimate fate of this patient. The presence of metastatic disease from such a small primary lesion is a discouraging sign. Fortunately, however, the remaining lymph nodes were found on examination to be free of carcinoma and the surgical margins of the primary growth, after local re-excision in one area, were also clear and considered adequate.

The gingival mucosa is one of the less common sites for primary intraoral carcinoma. Tiecke and Bernier[6] in an analysis of 401 cases of intraoral carcinoma report that 49 or 12% of these tumors were considered primary on the alveolar mucosa. The mandibular molar region is most frequently involved and carcinomas of the maxillary anterior gingiva are relatively uncommon. As with other forms of intraoral carcinoma, carcinoma of the gingiva is considerably more frequent in men than in women and it is rare before age 40.[5] Pinsonneault and Gill[3] report that only 3.4% of 2402 cancers of the oral cavity treated over a 24 year period occurred in women. Russell,[4] however, more recently reports a 76% increase in the rate of oral cancer in women during the period 1945-49 while the rate for men decreased 28% as compared with the figures for 1935-39.

The 5 year survival rate for early lesions is reported to be about 40% while advanced lesions show less than a 20% 5 year survival.[2]

Although the etiologic factors in oral carcinoma are unknown, it is interesting to observe that this patient did not have oral leukoplakia, gross oral sepsis, ill-fitting prosthesis or any of the other factors so frequently observed in relation to gingival carcinoma. This patient does not use tobacco. The events in this case emphasize the desirability of microscopic examination of diseased tissues removed from the mouth. Admittedly the vast majority of such specimens examined will show only inflammatory or benign hyperplastic conditions and the microscopic diagnosis will be entirely compatible with the clinical impression. Boyle[1] reporting on the experiences with the biopsy service at the University of Pennsylvania diagnosed 34 carcinomas out of a total of 1268 specimens seen over a 5 year period. In the vast majority of the cases submitted, histologic examination confirmed the clinical impression. However, there were 5 cases of unsuspected malignancy which were discovered by microscopic examination.

The early detection of unsuspected malignancy may prove to be a life-saving measure. For this reason it is essential to make greater use of the histologic examination.

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Retention of Periodontal Packs

by LEONARD S. HIRSCHFELD,* BERNARD H. WASSERMAN,† *New York, N. Y.*

VARIOUS surgical procedures are now used almost universally in the treatment of periodontal disease. After most of these procedures, a cement dressing is placed over the wound area, remaining in place during the period of healing. The purposes of the pack are to reduce pain, to prevent regrowth of tissue, to reduce hemorrhage and to protect the healing surface from mechanical trauma. The pack may be required for from one to three weeks, depending upon the extent of the wound area and the course of postoperative healing. If any part of the wound is exposed by premature loss of a portion of the pack, the patient may suffer considerable pain and there may be excessive re-growth of tissue.

Surgical dressings have been made more stable by increasing the strength of the pack by incorporating asbestos fibers in the mix[1] and by placing cotton pledgets in strategic locations in the operated area.[2] Wade[3] inserted tufts of cotton through interproximal spaces, where they were available, producing a "wheat sheaf." A medium-thick paste of zinc oxide and eugenol was then applied upon and between the fibers. Acrylic stents have been used as pack retainers by a number of authors[4, 5, 6, 7] with a variety of modifications of design and retentive devices.

There are several common situations where loss of part or all of the dressing occurs frequently in spite of any precautions taken by the patient. In such cases, methods may be used to insure the retention of the entire pack. This increased security also makes it possible to reduce the bulk, discomfort and unsightly appearance of the pack.

The materials used to increase pack retention are all available in the dental office, and require a minimal amount of time and effort to apply. These include: 1. stainless steel wire; 2. dental floss; 3. self-curing acrylic; 4. adhesive tin-foil; 5. existing appliances, and 6. copper bands.

When a gingivoplasty is performed to reduce enlarged gingivae, the immediate postoperative contour often approximates the normal. The interdental papillae are generally not removed, and it is therefore not possible to lock the labial and lingual portions of the pack together by having the cement pass interproximally. In addition, in many cases the surgery is limited to the labial side and no pack is required on the linguàl or palatal side.

In such instances, a simple wire framework will hold a relatively small amount of pack securely (Fig. 1 a&b). The wire may be placed either before or after the gingivoplasty. If the procedure involves the anterior area, an 8-inch piece of .010 dead soft stainless steel wire is looped tightly around one cuspid and twisted just enough times to have the twisted section reach to the mesio-labial line angle of the other cuspid. It is looped about the latter tooth and the ends are cut fairly short and tucked gingivally. It is important that there be some slack in the twisted wire so that pack material can be inserted between the wire and the wound surface. Additional pack is placed over the entire wound surface so that the twisted wire is enveloped by the pack. The cuspid loops should be placed below the cervices of these teeth, keeping the twisted part at the cervical line.

If the teeth are mobile and require full wire ligation,[8] two of the interproximal wires can be left uncut and be twisted into a framework for retention of the pack. The ligature itself is too close to the teeth to be enveloped in the pack.

*Associate Clinical Professor of Dentistry, School of Dental and Oral Surgery, Faculty of Medicine, Columbia University.

†Instructor in Dentistry, School of Dental and Oral Surgery, Faculty of Medicine, Columbia University.

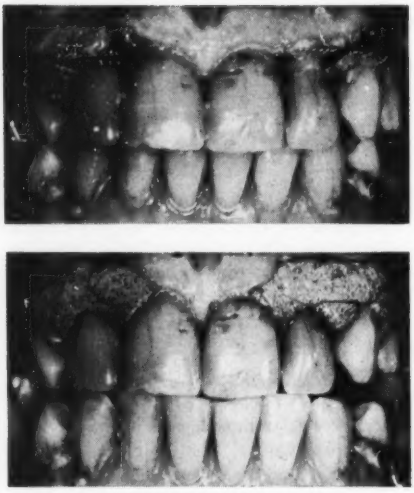
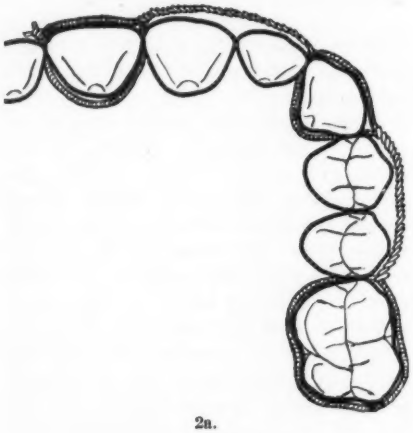
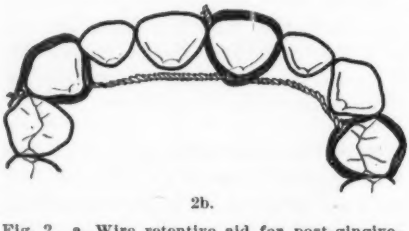


Fig. 1. a. Twisted stainless steel wire placed from cuspid to central incisor for pack retention. b. Surgical dressing in place over wire framework.

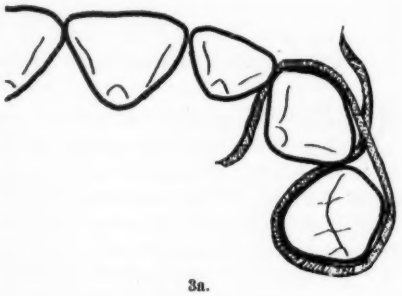


2a.

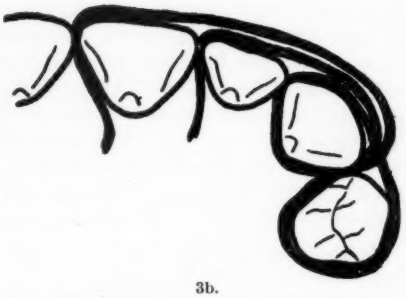


2b.

Fig. 2. a. Wire retentive aid for post-gingivoplasty pack showing loop around cuspid to improve stability. b. Lingual placement of wire pack retainer using additional tie on the central incisor.



3a.



3b.

Fig. 3. a. Initial steps in placement of dental floss retentive aid for dressing on gingivoplasty wound. b. Pattern of placement of dental floss for labial pack retention.

If the gingivoplasty is to include all teeth in a quadrant, the wire may be looped around the terminal molar, twisted until the cuspid is reached, looped around it and then extended to the opposite central incisor (Fig. 2a). This attachment in the middle increases the security of the framework, since the twisted sections lie loosely over the wound surface and would sag too far apically if not fixed in place by the cuspid. It is important to avoid sharp edges of wire, even though they are to be covered by pack, since the pack may be flattened out by action of the lips while it is still soft.

When a gingivoplasty or gingivectomy is to include lingual surfaces as well, an additional wire can be placed on the lingual side (Fig. 2b).

Waxed dental floss may also be used for a framework. If a scaffolding is desired just on the labial side of the anterior teeth, a length of floss is looped around the first

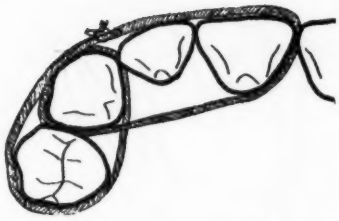


Fig. 4. Dental floss arrangement for labio-lingual retention.

5a.

5b.

5c.

Fig. 5. a., b. Stainless steel ligature wire arranged to produce a retentive loop for facial gingivectomy. c. Wire loop in place preceding gingivectomy.

bicuspid with both strands passing mesially to the labial of the cuspid (Fig. 3a). One strand is then passed around the cuspid from mesial to distal and then around to the labial of the lateral. The second strand is then passed around the lateral in a similar manner (Fig. 3b). This is continued until the terminal tooth involved in the surgery is reached and the two strands are knotted. Care should be taken to avoid impingement of the floss on the gingival papillae.

To produce retention both labially and lingually, the floss is looped about the cuspid and bicuspid. It is then passed between the two teeth and looped around the cuspid and the central or lateral incisor (Fig. 4). This pattern is continued until all the desired teeth are encircled. Between each two teeth, a strand of floss stands away from the teeth labial and lingual to the embrasures. Small pieces of pack are inserted into these embrasures and more pack is then placed on the wound area, enveloping the floss. In placing pack around floss or wires in interproximal areas, a cone of pack may be heated until it is soft and then pushed into place, using the cooler, harder base as a plunger.

A gingivectomy labial to one tooth, or distal to a terminal lower molar, presents problems of retention. One method for holding the pack is the placement of a copper band, which is fitted to the tooth. The band is prepared by making 3mm. slits lengthwise at 1mm. intervals around that part of the band which approximates the wound area. These fringes are then spread out to form a skirt which is then adapted to the gingival surface. The occlusal end of the band is trimmed just short of the morsal surface, and is closely adapted to the tooth. The dressing is then placed around the tooth and the band is forced into place. Additional dressing must be placed over the fringed gingival portion of the band to reduce the possibility of laceration of the cheek or tongue.

Placement of such a band may entail the necessity of forcing it through a tight contact point. A simpler method of retention is to construct a framework with .01-inch stainless steel wire. A loop is made about the cervix of the tooth, and the ends are twisted just enough times to form the proper size framework when the wires are again looped about the same tooth (Fig. 5 a, b, c).

When tissue is removed distal to an upper terminal molar, it is very difficult to retain a pack in the area. In fact, any gingivectomy on the palatal surfaces of upper teeth presents special problems. In order to

6a. 6b. 6c.

Fig. 6. a. Palatal tissue to be excised in gingivectomy, indicating palatal extent of wound area. b. Acrylic stent in position covering wound area and extending slightly onto normal tissue. Pack (P) is interposed between stent (S) and tissue. c. Acrylic stent in position showing relief holes in plastic for improved retention and ease of placement.

obtain contours which avoid a heavy ledge of tissue, a long bevel is usually required, extending towards the midline of the palate (Fig. 6a). When a pack covers this, the tongue compresses it before it is set, making the dressing very thin at the edges of the wound. Such fragile edges frequently crumble and the raw margin of the wound is exposed, resulting in considerable pain. This mishap, as well as complete loss of pack in the tuberosity area, can be prevented by the use of a thin self-curing acrylic stent.

A stent is easily made by a dental assistant, and requires very little chair time to apply. The proposed wound area is outlined on a study model, and a thin layer of acrylic is flowed or painted on, after the area has been tin-foiled. The stent extends somewhat past the edge of the wound and onto the palatal surfaces of the teeth, although not far enough to interfere with the occlusion. (Fig. 6b). Holes are made with a bur opposite the inter-dental spaces to permit wiring the stent to one or two teeth. If three or four teeth are involved, the stent is wired to a middle tooth. If five are covered, the stent is attached to the tooth at each end. It is important to have the holes fairly large so that there will be no difficulty in finding the interproximal space with the wire in case of a low contact point between the teeth. A shallow groove may be cut between the two holes for the wire to lie in, to prevent irritation of the tongue. Holes may also be cut into the stent to permit escape of excess pack material (Fig. 6c). The packing is applied and

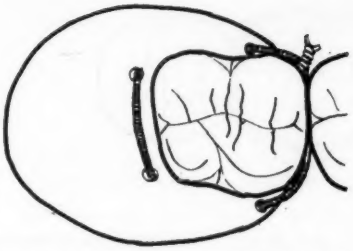


Fig. 7. Stent and ligation pattern for protection of wound distal to terminal tooth.

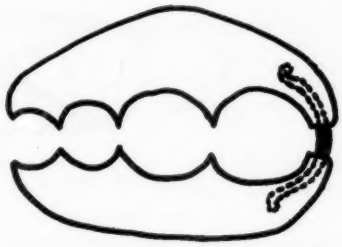


Fig. 8. Labio-lingual stent connected by .036 arch wire for total pack protection.

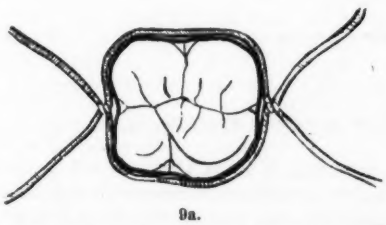
the stent is then wired in place. The wires are passed through from the palatal side, with the stent being held away from the pack until the wires are brought through. The stent is then pulled tightly into place, and the wires are twisted together on the buccal side. The twist is usually placed at the mesiobuccal line angle. The wires are then cut short and tucked interproximally.

When the stent is to be limited to the distal surface of an upper molar, four holes are made, as indicated in Fig. 7. The wire is put through the holes before the dressing is placed. When the stent is in position, the wire encircles the tooth.

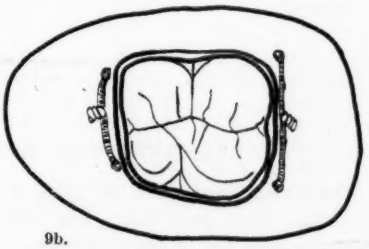
Retention on the labial or buccal side is generally not required, but when it is necessary, the stents on the labial and lingual surface may be joined by a stainless steel wire (.036-inch) which extends from the buccal to the lingual sections behind the terminal tooth (Fig. 8). The ends of this wire are bent into loops, which are embedded in the sections of the acrylic stent. The anterior ends of the stent are then perforated and a .010-inch wire is passed interproximally through the holes in order to wire the stent in place after the pack has been put into position.

When an isolated tooth is to have a gingivectomy, a stent may be made extending all around the tooth. Four holes are made in the stent, two mesial and two distal to the tooth, with a recessed trench between each pair. Before the pack is applied, two wires are twisted together and looped below the cervix of the tooth (Fig. 9a). They are twisted two or three times to secure them there, and the ends then are passed through the four holes. The pack is placed, and the stent moved down along the wires into position. The wires are then twisted together to fix the stent in place (Fig. 9b). This procedure takes less time to do than to describe, and avoids unpleasant emergency visits. A copper band may also be used in this situation, but it does not protect the edges of the wound.

If a wound extends further than originally estimated, the stent may not cover it



9a.



9b.

Fig. 9. a. Two-wire loop around isolated tooth requiring gingivectomy. b. Acrylic stent in position retained by double ligation.

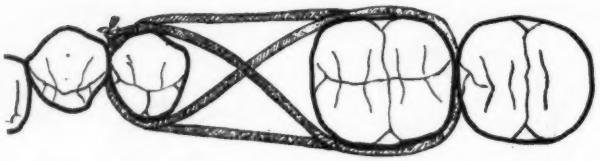


Fig. 10. Dental floss pack retainer utilizable over root, or edentulous area.

completely. In such a case, tin-foil is placed over the pack, extending past the edge of the wound and up under the stent, before the stent is wired on. If desired, additional acrylic may then be painted over the tin-foil, merging with the stent, protecting the edge of the wound completely.

The stent is removed with each pack change, scrubbed and replaced. It is important before wiring it to make certain that the stent does not interfere with the occlusion, since such pressure upon the wound can be very painful.

If it is desired to remove gingivae around a root in order to make a crown preparation possible, a stent can be used, or tin-foil can be placed over the pack, which is then retained by dental floss strung between the adjacent teeth (Fig. 10). The same arrangement can be used to cover a wound involving an edentulous area between two teeth.

When a partial denture is present, it can be used to retain the pack. Tin-foil is placed over the pack before the denture is seated. There must always be foil between the pack and the denture to prevent adhesion of the pack to the denture. After the denture is completely seated, all excess pack is removed and the denture is removed and reseated several times.

If the wound extends slightly beyond the denture, the tin-foil can protect the edge. If a large area is unprotected, a stent can be made to cover that area, designed so that it will not interfere with the denture.

SUMMARY

Various techniques for retention of periodontal packs under difficult circumstances have been described. Acrylic, wire, dental floss and tin-foil and copper bands have been used in various combinations.

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ASSOCIATION OF AMERICAN WOMEN DENTISTS

The annual meeting of the Association of American Women Dentists will be held in Dallas, Texas on Monday, November 10th, 1958, with headquarters at the Baker Hotel. A suite will be open on Sunday, November 9th, for visitors. For further information please write Stella Risser, D.D.S., 721 Medical Arts Bldg., Houston 2, Texas.

Effects of Drinking Metallic Salt Solutions on Alveolar Crest Resorption in the Syrian Hamster

by JOEL WISOTZKY* AND JOHN W. HEIN,** *Rochester, New York*

INTEREST in testing solutions containing metallic ions for their effect on the alveolar crest resorption in the Syrian hamster arises from reports in the literature that the chronic ingestion of certain ions of heavy metals such as lead, silver, and mercury sometimes causes the appearance of these metals in gingival tissue in the form of precipitated metallic lines. Logically, if certain metallic ions can accumulate in gingival tissue, it is natural to wonder what effect this will have on the periodontium. We know that certain metallic ions are important co-factors needed by some enzymes for effective catalytic activity. Contrariwise, other metallic ions, such as those of silver, cadmium, and gold, are capable of interfering with many enzyme systems that require a free reacting sulfhydryl group. The changing architecture of the fibrous attachment between cementum and bone is a dynamic process and also a vital process if the tooth is to adapt itself to changing stresses. Obviously, such a dynamic process requires active enzyme systems and these enzyme systems can be influenced by inhibitors or activators if exposed to their effects.

This paper is solely concerned with the effects of metallic ions on alveolar crest resorption, a process that can accurately be measured and recorded in jaw bones stripped of all soft tissue.

Costich[1] had suggested that the mandibular lingual surfaces may be useful as an area for screening alveolar crest resorption in the hamster, since these surfaces usually show the greatest involvement. Keyes[2] made measurements tending to confirm the impression that resorption of the alveolar bone in the hamster occurs in a bilaterally symmetrical pattern and also noted that maxillary and mandibular scores show good correlation, with scores slightly higher in the mandible.

METHODS

After trying various methods, the following method was adopted which appeared to be sensitive, reproducible, required the minimal amount of personal decisions, needed no drawing skill, and made possible a range of numerical values with a good spread between maximal and minimal degrees of intensity of resorption.

The lower left mandible, stripped of all soft tissue, was gently pressed into the surface of a small plasteline cube, which was mounted on the stage of a binocular dissecting microscope. The buccal aspect was on the surface of the plasteline and the lingual surface was facing upward and illuminated with a small fluorescent lamp. The modelling properties of the plasteline permitted a positioning of the lingual aspect so that it laid reasonably flat. A micrometer eye piece was calibrated by means of a Whipple eye piece disc and it was established that 155 units on the micrometer eye piece scale was equal to a distance of 1 millimeter on the surface being magnified at the power utilized.

Both eye piece lines were positioned directly on the jaws under each molar cusp at the level of the cementino-enamel junction. Then the movable line was moved down the exposed root to the point where bone coverage was present. The numerical value in terms of units on the micrometer was recorded from the six molar lingual root surfaces of the left half of the mandible.

A series of preliminary trials to check the accuracy of the numerical values in expressing the obvious state of generalized alveolar crest resorption, and also

From the Department of Pathology and Division of Dental Research, School of Medicine and Dentistry, University of Rochester, Rochester, New York.

*Joel Wisotzky was a United States Public Health Service Postdoctorate Research Fellow of the National Institute of Dental Research at the time this work was done.

This paper is based on a portion of a thesis submitted to the University of Rochester, New York, in partial fulfillment of requirements for the Ph.D. degree in the Department of Pathology.

**Present address: Division of Dental Medicine, Research and Development Department, Colgate-Palmolive Company, located at and in cooperation with Rutgers University, University Heights, New Brunswick, New Jersey.

Figure 1* shows a mandible with the lowest score of 0.00.

Figure 2 shows a mandible with a periodontal score of 50.25 which is close to the mean of the range encountered when scoring 234 hamsters' jaws by the method described.

to ascertain the reproducibility of the method, revealed the following:

1. The mesial root of the lower first molar was a particularly bad point of reference insofar as the accuracy of the method was concerned because the mesial surface of this root may often show an extensive area of vertical recession of the alveolar crest not correlated to generalized alveolar resorption. Many other students of the hamster periodontium have also observed this fact. Because this mesial resorption often extends onto the lingual aspect of the root, thus becoming involved in the scoring and tending to misrepresent the general status of alveolar crest resorption in the animal, it was decided not to include this reference point in the average.

2. The buccal and lingual plates covering the distal root of the third molar often fractured during the process of separating the lower jaw from the rest of the head and thus this point was regarded as an unstable point of reference.

Nevertheless, the bone resorption on all six root surfaces were always measured so that a record would be available. However, the first and last roots in the lower dentition were not averaged in the final periodontal score.

Scoring a group of eleven animals on two different days showed that the average difference between the scoring attempts was 3.84 units, the largest error was 9.50 units.

Considering that the mean of the platinum group will be shown to be 90.92 units and individual scores will run as high as 132.83 units, both the reproducibility and numerical spread afforded by this method appear to be satisfactory.

Twenty-five to thirty-five day old hamsters were distributed so that every animal in an experimental group had a littermate in a control group. The experimental groups drank the salt solutions *ad libitum* as their only source of fluid. Control groups drank distilled water *ad libitum*. The diet for both control and experimental groups was a cariogenic diet of the following composition:

Whole Milk (Powdered)	— 30%
Whole Wheat Flour	— 30%
Powdered Alfalfa	— 4%
Sucrose	— 35%
Na Cl	— 1%

The group size varied from 10 to 12 animals and they were started at 25-29 days of age except for the group that drank potassium acetate and their control; these latter animals were 32-35 days at the start of the experiment.

One of the foremost considerations in the selection of the metallic salts was to obtain soluble salts which upon solution and ionization would yield metallic ions distributed at various levels in the Electromotive Series. It was felt that since the Electromotive Series provides such useful information regarding the chemical behavior of a metallic ion, a selection of metallic ions distributed at different levels in the Electromotive Series might be particularly useful in gaining insight into the mechanism of action, should

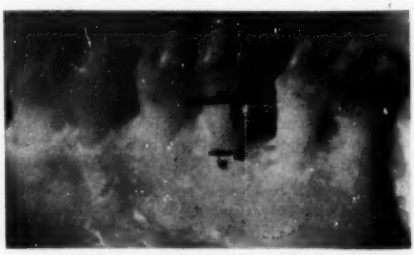


Figure 3 shows the mandible with the highest periodontal score of 132.83. This animal drank a platinic chloride salt 1 meq./1 for 124 days. Note the drifting of the 1st and 2nd molar teeth; also the darkness of the jaw bone. The distance between point A and point B was measured with the micrometer eye piece.

*Figures 1, 2, and 3 are photographs of the lingual surface of the mandible taken through the binocular microscope at 15x. The periodontal score for each jaw is given in order to aid in establishing a visual image of the state of alveolar bone resorption from the value of the periodontal score.

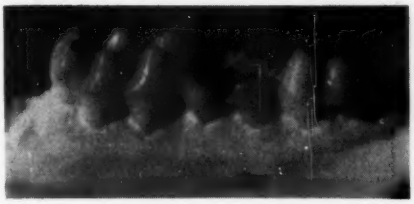


Figure 4* shows a quadrant in the upper jaw (15x) of the same animal whose lower jaw is seen in figure 1 and whose periodontal score was 0.00.

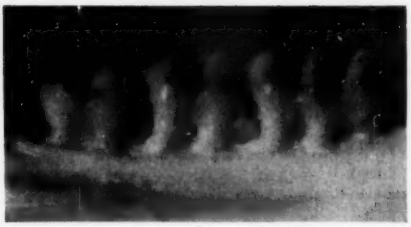


Figure 5 shows a quadrant in the upper jaw (15x) of the same animal whose lower jaw is seen in figure 2 and whose periodontal score was 50.25.

the metallic ions show any pronounced and significant effect upon alveolar crest resorption.

The following salts were administered in the drinking water to Syrian hamsters to determine their effects on alveolar crest resorption: Li_2SO_4, CH_3COOK, $KMnO_4$, $Mg\ SO_4$, $Cd\ SO_4$, $(CH_3COO)_2Pb$, V_2O_5, $Pt\ Cl_4$, $Pd\ Cl_2$, $HAuCl_4$.

RESULTS

A series of photographs are included with corresponding periodontal scores clearly indicated, Figures 1-6. This is to aid in establishing a visual image of the state of alveolar bone resorption corresponding to various periodontal scores. A photograph to illustrate the error which could be introduced by including the mesial root score has also been included, Figure 7.

Figure 8 presents a scatter diagram showing the periodontal scores of the animals serving as controls for those animals on the experimental solutions. The number of days that these controls were kept on the experimental diet is indicated on the abscissa. A bar indicates the average periodontal score for each group. Note the range of scores from values of 5 to values of over 110. The control animals ate the same diet and drank distilled water *ad libitum*, but subsisted on this forementioned diet from periods varying from 84 to 140 days. It is evident that there is a general tendency for animals kept on the experimental diet for longer periods of time to develop higher periodontal scores.

Table I summarizes the results of the experiments testing the effects of the various metallic ions on the alveolar crest resorption and comparing each experimental group to littermate controls. In certain instances a metallic ion was tested both in female and male hamsters, each having its own control. Whenever the experimental group average showed an obvious difference from the control, statistics are included to ascertain the probable significance of this difference. Students "t" test with Bessel correction was employed for statistical evaluation of mean differences between experimental and control groups. A value greater than 2 was considered as indicative of a probable change which was significant.

Figure 6 shows a quadrant in the upper jaw of the animal which had the highest periodontal score and whose lower jaw can be seen in figure 3. Note the drifting of molars in the upper jaw similar to that noted in the lower jaw. Note the darkness of the bone color in the upper jaw similar to that noted in the lower jaw. The platinic chloride solution appeared to affect bone color and density in the bones of the jaw and skull.

*Figures 4, 5 and 6 are photographs of the upper jaws, taken through the binocular microscope at 15x, of the same animals whose lower jaws were shown in figures 1, 2, and 3. The purpose of these three figures is to illustrate that measurements taken from the lower jaws give a reasonably good indication of the resorption in upper jaws.

THE EFFECTS OF METALLIC SALT SOLUTIONS ON ALVEOLAR CREST RESORPTION

Table No. 1

Salt Tested	Conc. of Metallic Component of Salt in Sol. meq./l	pH of Sol.	Number of Animals Scored	Days on Sol.	Average Score For Group	Stand. Error	"t"
$Li_2SO_4.H_2O$	1 meq.	6.20	11 ♂	84	19		
Control	H_2O	—	10 ♂	84	20		
$MgSO_4.7H_2O$	1 meq.	6.25	11 ♂	84	21		
Control	H_2O	—	10 ♂	84	20		
$CdSO_4$	¼-½ meq.	5.80	10 ♂	84	17	± 6.1	1.8
Control	H_2O	—	10 ♂	84	32	± 6.1	
$Pb(C_2H_3O_2)_2.3H_2O$	½ meq.	5.85	11 ♂	84	45	± 9.1	1.0
Control	H_2O	—	10 ♂	84	32	± 9.5	
V_2O_5	1 meq.	6.55	9 ♂	84	24		
Control	H_2O	—	11 ♂	84	20		
$KMnO_4$	1 meq. (MnO_4^-)	6.90	11 ♂	84	20		
Control	H_2O	—	10 ♂	84	20		
$Li_2SO_4.H_2O$	1 meq.	6.20	12 ♀	98	33		
Control	H_2O	—	7 ♀	98	46		
$MgSO_4.7H_2O$	1 meq.	6.25	10 ♀	98	35		
Control	H_2O	—	10 ♀	98	30		

*Brought to pH with .1N NaOH

continued Table No. 1

Salt Tested	Conc. of Metallic Component of Salt in Sol. meq./l	pH of Sol.	Number of animals Scored	Days on Sol.	Average Score For Group	Stand. Error	"t"
$CdSO_4$	¼-½ meq.	5.80	6 ♀	98	35		
Control	H_2O	—	6 ♀	98	43		
$Pb(C_2H_3O_2)_2.3H_2O$	½ meq.	5.85	8 ♀	98	36		
Control	H_2O	—	6 ♀	98	43		
V_2O_5	1 meq.	6.55	10 ♀	98	30		
Control	H_2O	—	8 ♀	98	30		
$HAuCl_4.3H_2O$	½ meq.	5.0*	10 ♂	98	35		
Control	H_2O	—	12 ♂	98	39		
$PtCl_4.2HCl.6H_2O$	1 meq.	5.5*	12 ♂	124	91	± 8.2	2.5
Control	H_2O	—	10 ♂	124	62	± 8.5	
$PdCl_2.2H_2O$	1 meq.	5.5*	10 ♂	124	59		
Control	H_2O	—	10 ♂	124	62		
$KC_2H_3O_2$	1 meq. K^+		9 ♀	140	58	± 9.9	1.3
Control	H_2O		9 ♀	140	76	± 9.9	

*Brought to pH with .1N NaOH

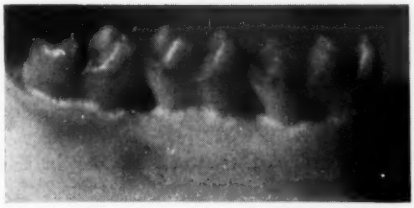
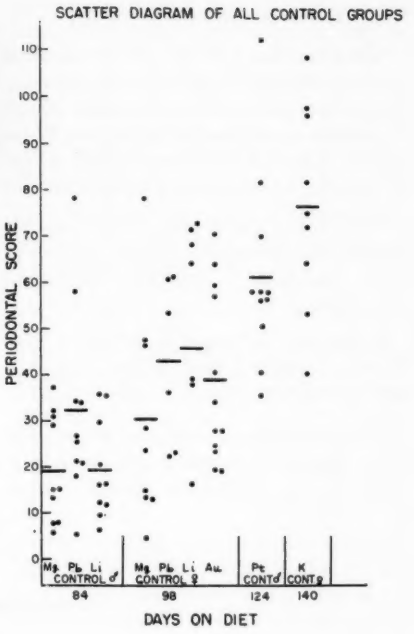


Figure 7 illustrates the error which can be incurred by including the measurement of the alveolar crest resorption from the mesial root of the lower first molar. Note that the large crest resorption on the mesial root does not reflect the crest resorption in the other roots. Alveolar bone resorption on the mesial root of the first molar may be due to local pressures arising from food packing and tongue positioning and in no way be associated with the general state of health of the tooth supporting apparatus.



The results clearly indicate that the majority of the metallic ions in the drinking solutions at the concentration tested had little effect on the resorption of alveolar bone about the roots of the molar teeth. Only the platinic chloride salt caused a pronounced acceleration of alveolar crest resorption. It is of interest that in the animals drinking the platinic salt the bones of the entire skull exhibited porosity, lack of density and lack of whiteness. An example of this can be seen by comparing the jaw bone in Figure 3, which comes from a platinum animal, with that of Figures 1 and 2 which are jaw bones from non-platinum drinking animals. Also by comparing the appearance of the upper jaw in Figure 6 taken from a platinum animal with Figures 4 and 5 from non-platinum animals.

It should be pointed out that some of the metallic ions affected the caries incidence at the same concentration and yet had little effect on the alveolar crest resorption. It should also be noted at this point that the periodontal score is not necessarily related to the caries experience in the animals. For example, animals on gold chloride experienced a low caries incidence while their controls had an active caries rate, yet the periodontal scores of both groups were reasonably close.

The cadmium ion which proved to be toxic at this level, causing a severe anemia and other blood abnormalities, had little effect upon alveolar crest resorption. Certain metals that gave some evidence of precipitating in the gingival tissue, such as palladium, cadmium and lead, did not significantly affect the alveolar crest resorption.

The accelerating effect of the platinic ion on bone resorption may prove of practical importance, for research in periodontal disease has been seriously handicapped due to the difficulty in causing this disease in some experimental animals. In order to induce periodontal disease in resistant animals, mechanical devices to irritate and apply pressure upon the periodontium (such as ligatures and rubber bands) have been tried. A suggestion arises from these experimental results that it may be possible to utilize the accelerating effect of the platinic salt to produce periodontal disease in animals heretofore resistant to attempts to induce this disease process.

SUMMARY AND CONCLUSIONS

The following salts of metallic elements were administered in the drinking water to Syrian hamsters to determine their effects on alveolar crest resorption: Li_2SO_4, CH_3COOK, $KMnO_4$, $Mg\ SO_4$, $Cd\ SO_4$, $(CH_3COO)_2Pb$, V_2O_5, $Pt\ Cl_4$, $Pd\ Cl_2$, $HAuCl_4$.

Of the ions tested, only the platinic ion caused any appreciable effect on alveolar crest resorption, it markedly accelerated the process.

Palladium, cadmium and lead caused metallic deposits in the gingival tissues yet did not affect the alveolar crest resorption.

While cadmium caused a severe anemia, other abnormal changes in the blood picture, and in general manifested a toxic effect on the animals, it did not affect the alveolar crest.

Except for the platinic ion, the number of days on the experimental diet was more of a factor in crest resorption than the incorporation of the metallic ions in the drinking solution.

Finally, the position of a metallic ion in the Electromotive Series does not correlate with its effects on alveolar crest resorption.

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Conclusions of the Temporomandibular Joint Clinic at Columbia*

by LASZLO SCHWARTZ, D.D.S†

FROM the observations made during the past ten years at the Temporomandibular Joint Clinic at Columbia University it is possible to formulate some conclusions. These are the following:

1. In the over one thousand patients examined, the symptoms found were not those emphasized by Costen; nor did the symptoms comprise the complex described by him. Where individual symptoms of Costen's syndrome were met, we were unable to relate them to "bite closure."

2. We were unable to relate symptoms associated with the temporomandibular joint to any "excessive" range of condylar movement (usually considered to be anterior to the crest of the articular eminence). As had already been pointed out by some earlier investigators, we found that movements considered "abnormal" according to some arbitrary geometric norm, were entirely physiological for many individuals. In our opinion, the use of sclerosing agents, intra-articular or periarticular, has no physiological basis. Many patients gave histories of bad reactions to such treatment and degenerative changes were found in the joints of a few.

3. We did find in the temporomandibular joint, organic disorders common to all joints. The most common organic disorder was degenerative joint disease. Found less often were rheumatic disease other than

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†Clinical Professor of Dentistry and Director, Division of Clinical Oral Physiology, School of Dental and Oral Surgery, Columbia University.

degenerative joint disease, fractures, and neoplasms. The incidence of organic disease was small.

4. The disorder involving the temporomandibular joint found most often was a pain-dysfunction syndrome. The most usual signs and symptoms of this syndrome are similar to ones affecting other joints of the body—a constant dull ache with limited movement, aggravated by function.

5. The pain-dysfunction syndrome often appears first in the form of clicking, subluxation, or dislocation. Incoordination of the mandibular muscles is believed responsible for these symptoms. Subluxation was the name given, for the lack of a better one, to the condition of the jaw slipping momentarily or the teeth sometimes not meshing.

6. The incoordination symptoms are often followed by painful limited mandibular movements caused by spasm of some of the masticatory muscles. When spontaneous remission does not occur and when spasm is not relieved by treatment, such spasm may become cyclical. A self-perpetuating feedback mechanism is believed to be responsible. If such dysfunction is of long duration, contracture of the masticatory muscles, with limited though painless mandibular movement, may occur.

7. Whether the spasm of the masticatory muscles follows painful impulses arising in the joint, or whether such impulses arise directly in the muscles themselves, is not known. It seems likely that both mechanisms play a part.

8. No single cause exists for the pain-dysfunction syndrome. A constellation of etiological factors, including occlusion, seems to be responsible. What the patient does with his occlusion in reacting to stress seems to be more important than the malocclusion itself. Malocclusion by mechanically increasing the amount of force or altering its direction can make the chance of injury more likely. More important than the type of malocclusion, however, is the amount and kind of muscular activity together with the reaction of the individual patient to such activity. Changing an existing occlusion, by either grinding or restoration, was frequently associated with the onset of the pain-dysfunction syndrome. Change in proprioception, no matter how slight, seems to be more important than a long standing malocclusion, no matter how irregular.

9. A hypothesis is offered for consideration and trial. This is based on the fact that with stress, the tension of skeletal muscle increases. When such increased tension occurs in the masticatory muscles for long periods, four general types of reactions have been observed. In some individuals the consequence is muscle hypertrophy, tooth wear, and hardening of alveolar bone; in others periodontal disease; and in still others, the pain-dysfunction syndrome or degenerative joint disease. As a result of dental treatment, the individual response may change. For example, in some patients, splinting of loose teeth with fixed bridges precipitates muscle spasm with either limitation or subluxation symptoms.

10. To test this hypothesis, a procedure for the physical examination of the functioning of the jaws, joints, and musculature has been developed. Such examination, in addition to broadening the scope of diagnosis, should help standardize our methods of obtaining clinical data. This should make it easier for clinical investigators to communicate with each other. A complete physical examination of the masticatory (or stomatognathic) system would also serve to place electromyographic and roentgenographic findings in truer perspective. Roentgenography is a useful adjunct in diagnosis but must, of course, be subordinated to the history and physical examination. One must be on guard against any geometric evaluation of condyle-fossa relationships if for no other reason than that a roentgenogram is a distorted static two-dimensional shadow picture of the hard

structures of a complex three-dimensional moving joint.

11. We believe that within many specialities of medicine, particularly physical medicine and psychiatry, can be found knowledge and technics which would make the management of temporomandibular joint disorders more effective. Some of these have been presented. More work along these lines should make us less dependent upon altering occlusion as the only way to treat temporomandibular joint disorders. We should be particularly wary of any rigid, mechanical concept of the norm inherited from the full denture period of American dentistry. A broader approach to the problem of disorders of the temporomandibular joint cannot but help free us from the belief that the occlusion of the teeth is always stable and cannot change in the same individual from one moment to the next. Our investigations have demonstrated that though "prematurities" or "occlusal interferences" may contribute to the onset of muscle spasm, muscle spasm can and does create "prematurities." Our research has made us much more hesitant in treatment involving occlusion. We feel that it should not be undertaken until both the patient and his muscles are relaxed. We also feel, through our collaboration with Dr. Ruth Moulton, that in some patients occlusion should not be touched at all. With Dr. Moulton's help we are learning to view the whole patient and if as a result we feel a reservation, we do not brush it aside just because we see an occlusion that offends our sense of what is "normal."

12. It is our belief that many conditions labeled disorders of the temporomandibular joint are not temporomandibular joint disorders at all, but rather muscle dysfunction. However, until greater knowledge as to what really goes on in disorders of the temporomandibular joint is at hand, we should proceed cautiously. By the cilnical study of dysfunction, correlated with basic research of the physiology of the masticatory system, we can work towards a better understanding of the physiological system that we treat. Towards this end, electromyography holds out much promise. In the meantime, we must rely upon hypotheses. But such hypotheses should be based on observation and such scientific knowledge as is available. They need not presume the irreversible or pathology when the reversible or physiology will do. Sound hypotheses are more than wild guesses. Based on observations they lead to further observations. Hypotheses must, of course, be freely altered or readily discarded, as new facts appear.

But, in the meantime, what should be done with the patient before us? We should be guided by an aphorism of Hippocrates. This reads that, "Extreme remedies are very appropriate for extreme diseases." Temporomandibular joint disorders are rarely extreme. In our opinion, it is sound management to observe carefully, proceed cautiously and to be on constant guard so that even if we cannot help our patient, we do not harm him.—*630 West 168 Street, New York 32, New York.*

ORAL PATHOLOGY CERTIFICATION EXAMINATION

The American Board of Oral Pathology will give its annual examination for certification on December 6 and 7, 1958. The place of the examination will be announced when the geographical location of the applicants has been determined. Information relative to certification can be obtained from the secretary.

All applications should be completed and forwarded to the secretary, Donald A. Kerr, D.D.S., University of Michigan School of Dentistry, Ann Arbor, Michigan, at once.

A Desquamative Cytological Study of the Gingivae and Oral Mucosae in Women

by J. R. TROTT, B.D.S.,* *London, England*

DESQUAMATIVE cytological and biopsy technique studies have revealed that cyclical changes occur in the hornification of the vaginal epithelium (Papanicolaou, 1933; Cruickshank and Sharman, 1934; Rakoff, Feo and Goldstein, 1944). The rhythmical variations of the oestrogen concentration controlling the menstrual cycle produce these cyclic changes in the vagina and although some workers believe the hormonal action to be indirect in nature (Cruickshank and Sharman, 1934), there is general agreement that the phases of the menstrual cycle can be studied by changes in the hornification of the vaginal epithelium.

Similarities exist between the oral and vaginal epithelia which have led many workers to believe that the influence of the sex hormones on each may be very much alike (Ziskin, Kamen and Kittay, 1943; Ziskin and Moulton, 1948; Iusem, 1950). Not all workers agree on this (Papic and Glickman, 1950; Montgomery, 1951), and it was therefore decided to repeat the work on a greater number of women over a longer period of time than had been done before.

MATERIAL

It was felt that if there was a variation in the keratinisation of the oral mucosa or gingiva, related to the menstrual cycle, then a number of women with relatively regular and normal menstrual cycles should be studied over a series of consecutive cycles.

Eleven female patients ranging in age from 19 to 28 years of age were examined twice weekly through several menstrual cycles. The number of days in each individual's menstrual cycle can be seen from Table I, whilst the number of consecutive cycles observed from Table II. The total number of cycles that were studied amounted to thirty-nine, whilst the total number of smears examined were 738. The reason for this discrepancy is that at the beginning of the study it was decided to record the commencing of each menstrual cycle in five of the subjects, whilst the remaining six would keep their own records and after the author had tried to work out the cyclic pattern, if any, they would then let him have the exact date of commencement of each menstrual period. In doing this it meant that there was a certain degree of overlap and for the purposes of this study only completed consecutive menstrual periods are reported.

None of the women were under treatment from their physician for any menstrual disorder and it will be noted from Table I with the exception of subjects Nos. 3, 4, 9 and 10 the menstrual cycles were reasonably constant in length. The women under observation could therefore be said to be as near normal with regard to their menstrual cycle as it is possible to get in any group of women.

TABLE I

No.	*Age*	*Marital Status*	*Number of Days Between the Commencement of Each Menstrual Period*
1	23	Single	28, 27, 28, 29, 28.
2	20	Single	29, 27, 27, 27.
3	19	Single	28, 26, 30, 33, 26.
4	23	Married	27, 34, 31.
5	23	Single	29, 27, 28.
6	28	Single	28, 28, 29.
7	27	Single	28, 31, 32.
8	20	Single	26, 27, 29, 28.
9	21	Single	33, 28, 23.
10	20	Single	33, 34, 25.
11	25	Single	29, 31, 33.

Table I shows the age, marital status, and the number of days between the commencement of each individual's menstrual period in the eleven female patients examined.

No lesions were observed in the oral mucosa during the period of study and the surface characteristics of the gingiva, both marginal, papillary and attached, in the anterior regions could be considered as normal with regard to the colour, contour, and consistency (Trott and Wade, 1954.) Oral hygiene methods as carried out normally by each individual were continued throughout the period of study. Only one person had to be excluded from the in-

*General Dental Surgeon, Department of Periodontology, Postgraduate Medical Federation, Institute of Dental Surgery, Eastman Dental Hospital, University of London.

TABLE II.

Number of cycles	5	4	3
Number of subjects	2	2	7
Total number of cycles observed.	39		

Table II shows the number of consecutive menstrual cycles observed and the number of subjects for whom this was possible.

TABLE III.

Group	*Percentage of Keratinised Cells Present*
A	30%
B	50%
C	70%
D	90%

Each smear following staining was classified under one of the above four groups.

vestigation due to a severe cold, but this was only after three consecutive cycles had been recorded. No account was kept of the incidence of colds, but they were infrequent and would not have appeared to influence the findings. Thus oral and gingival films were obtained twice weekly from three to five months as indicated in Table II.

METHOD

Films were made from scrapings taken from the attached gingiva on the labial surface in the anterior mandibular region and from the adjacent vestibular mucosa. To obtain films these areas were scraped gently two or three times with a wooden spatula and the material transferred directly to a glass slide. To avoid the possibility of producing changes in the surface epithelium by too frequent scraping, the specimens were taken alternatively from the right and left sides of the mouth.

The films were fixed and stained according to the method of Shorr (1945) particular care being taken to fix the films while still moist, since drying so alters the straining reaction as to make the results unreliable (Fig. 1). The films were examined under a magnification of 63 diameter, and classified according to Table III. Four stages in the process of keratinisation of an epithelial cell can be recognized by this technique (Papic and Glickman, 1950).

1. Non-keratinised cells possess a nucleus and the cytoplasm stains green.

2. The earliest stage of keratinisation are indicated by blue staining of the cytoplasm, the nucleus still being present.

3. The cytoplasm of the partially keratinised cells stains pink, the nucleus still being present.

4. The completely keratinised cells stains pink, the nucleus now being lost.

An overall picture of the whole film was taken and graded as in Table III. Ideally it would be desirable to carry out an accurate differential count of the cells present in each smear but there are two practical difficulties. Firstly, the cells in a film tend to clump together, making it a practical impossibility to carry out a differential count. Secondly, the keratinised and non-keratinised cells were not always spread homogenously over the slide, and by counting one area a false picture may have been obtained.

The grading for each film was transferred to a graph on which the degree of keratinisation was plotted against the day of the menstrual cycle. The day upon which the menstrual flow commenced was considered as the first day of any particular cycle. In recording the gradings, on each day that the films were taken no reference was made to the graphs. The films were examined, the number of the subject noted and the grading recorded on a blank sheet of paper. On the completion of 22 mucosal and gingival examinations, the records were transferred to the graphs. It was hoped in this way that any possible bias would be eliminated.

RESULTS

The observations recorded on the graphs were analysed in three ways.

1. By a direct examination of the original recordings over the number of periods as shown in Table II. (Fig. 2.)

2. By making a composite graph for

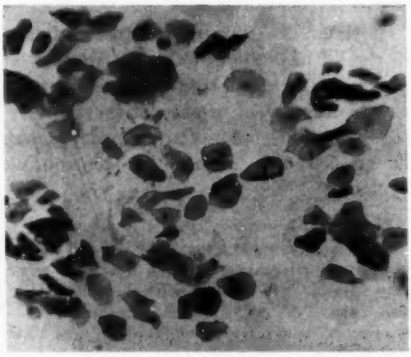

Fig. 1. A typical smear taken from the vestibular mucosa and stained by Shorr's method. The film shows a mixture of non-keratinised and fully keratinised cells. This smear would be graded as A magnification X110.

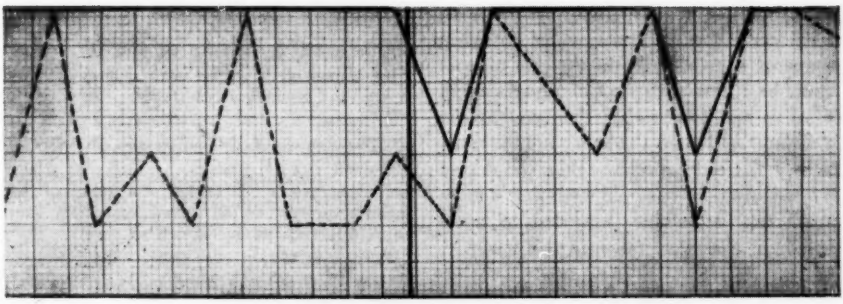

Fig. 2. A graph showing the variation in keratinisation. The continuous line shows the keratinisation of the attached gingiva, whilst the interrupted line shows the keratinisation of the vestibular mucosa. The readings were taken over two complete menstrual cycles.

each patient over the number of complete menstrual cycles observed. Every fourth day was taken in each cycle, an arithmetical mean obtained, and plotted on a single graph.

3. The total number of recordings observed in the 39 cycles were transferred from the original graphs to one composite graph. The abscissa representing the number of days in a cycle and the ordinate, the four degrees of keratinisation.

Graphs prepared as just indicated, showed no evidence of any cyclical changes in the degree of keratinisation in the oral mucosa. Nevertheless, changes do occur at different times in successive menstrual cycles in the same patient, showing sometimes a rapid alteration in pattern from nearly complete keratinisation to very few observable keratinised cells. The fluctuation from one extreme to the other may occur fairly frequently in one cycle whilst in the following, there may be a long period during which the degree of keratinisation, whether it be high or low remains constant. However, the frequency of fluctuation in the degree of keratinisation, in general is marked but it has not been possible to demonstrate any causative factor.

With regard to the graphs prepared of the degrees of keratinisation found on the attached gingiva. In most cases the films showed that there were at least 90% keratinised cells present. There were occasional fluctuations in the degree of keratinisation which showed no relation to the cyclic pattern of the menstrual cycle. The frequency of the fluctuations in the degree of keratinisation was less than that observed in the vestibular mucosa, and the changes in degree were less marked. Again, there seemed to be no apparent reason for the variations seen.

When the graphs of the degrees of keratinisation of the oral mucosa and the attached gingiva are compared there appears to be no relationship between the two.

DISCUSSION

There are limitations to the endurance of patients one chooses in such studies as this, yet it seemed better to observe a number of consecutive cycles in a few patients than to take a large number of patients for a limited period. Similarly one should ideally control such an experiment as outlined by taking vaginal films and early morning mouth temperatures, but here again one must remember the patient and if one has to rely upon a number of people taking accurate measurements for some length of time, the results may be open to doubt.

There is, however, little evidence to support the thesis that the oral mucosa shows cyclic changes related to the menstrual cycle. There is no doubt that continual changes in the pattern of keratinisation do occur as evidenced by the rapidly changing

points on the graphs that were kept. It seems likely that the continually changing degree of keratinisation in the oral mucosa would be due more to the functional stimuli of saliva and food rather than oestrogenic in nature. Although the Shorr stain is satisfactory for estimating roughly the degree of keratinisation, it would seem that a more satisfactory technique is needed to assess such films that were taken in this study.

SUMMARY

The desquamative cytology of the gingiva and oral mucosa was observed through a number of consecutive menstrual cycles in eleven normal women. No evidence was found of a cyclic pattern related to the menstrual cycle.

ACKNOWLEDGEMENTS

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Thoughts Concerning Cause, Pathogenesis, Treatment and Prevention of Periodontal Disease

by SUMTER ARNIM, B.A., D.D.S., PH.D.,* *Houston, Texas*

THE purpose of this essay is to present certain thoughts concerning the etiology and pathogenesis of periodontal disease with their clinical implications. These concepts stem from study of human autopsy specimens serially sectioned so that the periodontal lesion may be seen in its entirety. Hypotheses derived from the histologic studies have been modified from time to time as clinical therapeutic tests have been made in dental school clinics, personal private practice† and in the private practice of departmental colleagues. Certain contributions to the scientific literature,[17, 18, 27, 29, 31] phase contrast microscopy and cinemicrography have also provided data which calls for the formulation of current concepts derived from all the foregoing sources.‡ A discussion of these ideas follows:

THOUGHTS CONCERNING CAUSE

The direct cause of periodontal disease is the adherent colonized microbial mass found on the tooth surface at the gingival crest, in the sulcus and in the periodontal pocket with its associated bordering communities of motile and nonmotile bacteria, protozoa, leukocytes, drifting desquamated cells as well as other varieties of microscopic life, living or dead.[5, 10] They are enveloped and permeated by a thick, viscous, serous exudate that helps hold all these elements and their noxious end products in contact with the tooth and gum.[7] *This microscopic domain with its populace of microbial life, tissue desquamates and exudates will be called the periodontal microcosm in this essay.* As the communities of microbes within the microcosm grow in size the exudates grow in volume and are supplemented from time to time with hemorrhage from the gingival capillaries.[1]

In stressing the role of the adherent colonized masses and the bordering communities with their teeming microscopic populations, no effort is made to discount the importance of those predisposing factors which have been given prominence in dental literature. Nor is it intended to deny that some individuals have the microorganisms but not the disease. This is a separate immunological problem.

It is thought, nevertheless, that the microbial communities with their end products create a periodontal microcosm which, in its entirety, is of first importance as the cause of periodontal disease.[6, 8, 9] The disease does not occur without the colonized organisms. The lesions heal when the periodontal microcosm is removed completely and that part which grows back is removed each day.

THOUGHTS ON PATHOGENESIS

The pathogenesis of periodontal disease begins at the time the tooth erupts. The tooth is the stony foundation to which the

*Professor of Pathology and Director Postgraduate School of Dentistry, The University of Texas Dental Branch. This research was supported in part by U. S. Public Health Service Grants D-200 and D-389.

†Thanks are due those conscientious individuals who through their intelligent understanding and diligent application of principles and practice as described herein have taught the author that periodontal disease can be prevented and controlled by the cooperative patient.

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microbes adhere and on which they colonize while building their relatively permanent abodes. The organisms find this site congenial because there is a continuous outpouring of nutritious tissue fluids from the healthy mucous membranes and marginal gingiva that bathe the area constantly. A microscopic quantity of serous exudate seeps through the thin epithelium of the healthy gingiva adjacent the tooth surface and oozes outward into the oral cavity. It carries in its slow current desquamated epithelial cells and occasional vital leukocytes.[2] The exudate may be observed by gently pulling the margin of the gum away from the tooth and looking into the crevice where it forms sticky, weblike, viscous filaments between soft tissues and tooth. This outpouring of exudate supplies the adherent microbial communities with a constant, continuously renewed food supply.

As long as the connective tissue fibers in the free gingiva are abundant and healthy, the gingival cuff fits the neck of the tooth snugly. These connective tissues with the serous exudate and living leukocytes provide a physiologic seal which allows for limited movements of the gingiva. This dynamic anatomic arrangement at the neck of the tooth is constantly undergoing conformative changes. It adapts and readapts to the tooth surface during eruption, orthodontic movement, mastication or vigorous gum massage. It also regenerates following trauma from operative procedures, food particles, oral surgery or accidental injuries.[32] *It is only by means of this plastic contiguity of the marginal gingiva that the integrity of the healthy periodontal tissues is maintained by the physiologic seal as conditions alter around the neck of the tooth.*[25, 28, 33, 34]

As the exudate slowly oozes toward the colonized mass of microbes at the gingival margin, noxious products and microorganisms[3, 30] work their way apically between tooth and gingiva. Soluble toxins diffuse through the thin epithelium into adjacent connective tissues. The viscous exudate, in which the periodontal microcosm lives, also serves as a pathway for growth and extension of the microbial communities along the tooth surface toward the apex. As the periodontal microcosm develops on the tooth surface the first microscopic changes occur in the gingiva next to the organisms. These are proliferation and desquamation of epithelium, serous and cellular exudation and lysis of connective tissue fibers. Whenever and wherever the periodontal microcosm is found lesions are present. When no bacteria colonize no lesions are seen.

Ingested food plays a transient role as a source of nutrient for the microbes. Should food debris lodge within the periodontal microcosm it becomes useful to the bacterial populace as a nutrient only after it is converted to a soluble state. Recognizable food particles are seldom seen within the microcosm.

As the adherent microbial populace proliferates and achieves considerable volume adjacent the gingival margin, those organisms nearest the tooth surface and deepest in the organized colony may become infiltrated with mineral deposits forming calculus.[19, 20] The hard concretions are covered with a soft bacterial layer on their outer or gingival surface that may in turn mineralize as it achieves thickness and volume. Whether the communities of organisms calcify next to the tooth surface or not, they alter the topography of the area next to the gingiva and act as a barrier, like a reef at a river's mouth, to passage of the exudate. This barrier with its filamentous surface provides an eddy or backwash in which bacteria, protozoa, cells and other varieties of microscopic life writhe, squirm and toss about in teeming procreative confusion. As the individuals in this community age and die, the noxious end products formed penetrate adjacent tissues in increased quantities.

As a consequence, the gingival exudate again increases in volume, and many polymorphonuclear leukocytes pass through the epithelial layers working their way out between the epithelium and tooth surface

toward the colonized masses of microorganisms.[21] The thin, delicate layers of epithelium adjacent the tooth surface proliferate further sending deep, long extensions of the germinative layer into the fibrous connective tissues of the submucosa. Desquamation of epithelial cells is increased markedly. Soft, keratinous, cuticular films collect on tooth surfaces and on the surfaces of adherent bacterial masses. As the cellular and serous exudates first appear within the connective tissues of the marginal gingiva, lysis of the circular fibers occurs. With loss of the supporting connective tissue the gingiva loses its firmness, stands away from the tooth surface; the sulcus deepens, and the periodontal pocket begins.

The inhabitants of the periodontal microcosm find this tissue response ecologically favorable. It does not weaken the attachment of the adherent colonized masses to the tooth surface in any way. The flabbiness of the gingiva, with increase in exudation and desquamation, provides more room and nutrient for growth and development of the microorganisms.

As the epithelium along the dental slope of the marginal gingiva proliferates and sloughs, tiny capillaries extend through the surface into the periodontal microcosm. They enrich the media from time to time with hemorrhages, recognized by the patient as bleeding gums. All connective tissue fibers near the bacterial communities disintegrate and disappear. Those that are not close to the lesion remain intact.

The presence of a periodontal microcosm of such complexity affects the deeper tissues as well as those in the immediate vicinity. Alveolar bone, cementum and dentin are involved by resorption as the sulcus deepens into a periodontal pocket. When periodontal membranes disintegrate on one or more sides of the tooth, mobility is felt by the patient. Other symptoms described as "a strange consciousness of the teeth," "itching of the gums" and/or actual "pain" during mastication are common complaints.

From this stage on, lesions steadily grow progressively worse. Finally, the tooth becomes so loose that it is shed. The wound then heals and soft tissues again recover the health that was theirs prior to tooth eruption and colonization of the microbial masses. The periodontal microcosm that existed in the oral cavity because of its ability to adhere to the tooth surface within the path of a nutritious outpouring is lost with the tooth. All types of periodontal disease are said to respond favorably to extraction of the teeth.

THOUGHTS ON TREATMENT

Sir Arthur Keith,[15] in a book dealing with the science of orthopedics, wrote certain things of great interest which are applicable to problems of treatment in the field of periodontology. He said, "The surgical triumphs won since the discovery of anesthesia, antisepsis and asepsis have tended to make the surgeon forget that he is not a master but only a servant; the power to heal lies not in his hands but in the recuperative powers of his patient's body. He can assist the natural powers inherent in all living flesh but he cannot replace them. John Hunter devoted his life to prove that fact; those who read the medical lessons of the war aright realize that we have to return to Hunter's point of view. We can help the sick and the maimed in only one way . . . by aiding and abetting the natural defensive and recuperative mechanisms of the human body. Our methods must be based on a knowledge of these mechanisms." In addition he pointed out, "There is all the difference in the world between the pious enunciation of a principle and its strict, systematic, and complete application."

Principles of treatment are amenable to standardization, but specific technics should be custom-tailored for the individual dentist and by the dentist for the individual patient. Three principles are universally applicable in all periodontal treatment.

The first principle is useful as a directing influence during all operations performed

by the dentist. In a truly broad sense it might be termed the "S" or surgical principle. This principle is a valuable guide to the dentist during prophylaxis, curettage, gingivectomy, vestibular surgery, splinting, medication, dressings and all operations of a strictly professional nature. The "S" principle is: *Successful treatment depends on a strict, systematic and complete removal of the periodontal microcosm, in its entirety, by the dentist. Following removal of all injurious agents, healing is governed by the recuperative powers of the patient's body.*

The truth embodied within the "S" principle is well illustrated by that most radical of all treatment methods—extraction of the tooth. All will agree this is the sure, thorough way to remove all causative agents of periodontal disease. The operation also eliminates the colonization site of the microbes. Without a tooth organisms remaining in the mouth are even less well situated than a colony forming calculus in a tonsillar crypt. The community in the crypt is destroyed by desquamation, exudation and suppuration from all sides. In both cases the recuperative powers of the patient's body heal the lesions after the bacterial communities are removed.

The problem becomes a complex one when the aim of treatment is preservation of the tooth and elimination of the microcosm. This complexity coupled with the many variations found in the individual patients' bodies has led to the development of a great variety of technical methods for treatment, each satisfactory when it implements the governing "S" principle effectively. It is plain, common sense to try first the simplest and least traumatic of all suitable treatment methods. This therapeutic test should be thorough and the results evaluated with painstaking care before methods involving greater surgical risks are employed.

The second standardizing principle applicable in all periodontal treatment will be called the "E" for education principle. It is: *Final success of any treatment method depends on education of the patient by the dentist concerning the cause and prevention of periodontal disease.* It is an essential part of all periodontal treatment methods because the microcosm, following complete removal by the dentist, grows back abundantly within twenty-four hours. The adherent microorganisms begin to form calculus in a few days and the periodontal microcosm with its noxious products is soon re-established. Obviously, the patient is the only one who can remove the proliferating organisms as fast as they grow, so he must be taught how to get rid of the major part of the new growth each day.

Here again, satisfactory methods are multitudinous and each is successful just to the degree that it effectively implements the "E" principle. Best results are obtained when the dentist teaches the patient to recognize the periodontal microcosm on the teeth and to know that it is the cause of disease. The colonization sites and the path of extension into the underlying tissues are important. But most important of all is the knowledge that harmful microbes proliferate in countless numbers each day and, consequently, they must be removed regularly without fail.

The basic instruction is supplemented by teaching the patient correct use of toothbrush, dental floss and other aids for personal oral hygiene suitable to the individual case. When the periodontal microcosm occupies deep sulci or pockets, a water syringe, or similar device, is useful for washing out many of the microorganisms and noxious end products. It is common knowledge that the most skillful professional treatment fails to control periodontal disease unless the patient applies effectively the lessons learned concerning cause and prevention.

This fact has led to the elaboration of the third standardizing principle. It will be called principle "T" for testing. Principle "T" is: *The dentist and the patient*

must test the effectiveness of treatment and hygiene methods frequently to be certain that the cause of periodontal disease is removed adequately.

The dentist has several technics useful for this purpose. Gingival biopsy provides an exact and scientific evaluation of the status of the tissue.[12] The tissue is normal when epithelium and connective tissue are intact and desquamation and exudation are minimal. If there are microscopic signs of inflammation, the cause of periodontal disease is present and active.

Another valuable test is the study of teeth that are to be extracted in the near future. The dentist can operate on these teeth with any desired technic and following extraction the results of the operation may be evaluated with hand lens, dissecting microscope or histologic sections. This procedure is an excellent means for developing technical skill. "Hopeless" teeth, third molars and others suitable to the purpose are used. A simple hand lens is sufficient to reveal remaining adherent microorganisms, calculus, and irregularities of root surface. Testing end results realistically leads to marked improvements in surgical technics.

The least troublesome and most practical test is done with disclosing solution.[26] Basic fuchsin** is excellent for this purpose. The dentist uses it regularly to locate those zones where treatment methods have failed to eradicate the microcosm adequately. It is used also to show patients the periodontal microcosm and help them recognize colonization sites, the volume present and the significance of the microorganisms in relation to clinical signs of periodontal disease. The patient is instructed to test the effectiveness of his personal oral hygiene with disclosing solution at home. This saves time in the dental office. It also achieves the purpose faster as he soon learns to correct his own mistakes.

The object of personal oral hygiene is removal of the microorganisms. The disclosing solution tells the tale and is all revealing in this respect. Final responsibility for success of treatment rests with the patient only.[23] Microbial populations regenerate too quickly for the practitioner to assume this responsibility. The patient who uses a disclosing solution regularly knows when he is discharging the responsibility successfully. This is the beneficial result achieved when principle "T" is applied practically.

THOUGHTS ON PREVENTION

The salubrious benefits derived from a realistic program of personal oral hygiene (thorough removal of periodontal microcosm daily and regular check with disclosing solution as needed) are beyond simple factual description. Enthusiasm for the beneficial results obtained by conscientious patients prejudices the practitioner to such an extent that he tends to preach a gospel of hygiene rather than a lesson in prevention.

A discriminative literary search reveals that since 1450 A.D.[11] many authors have learned periodontal disease can be prevented by the educated patient. There is a common element present in their writings, namely, enthusiasm for the subject, and a general agreement in their recommended procedures.[13, 14, 16, 22] The oft repeated theme concerning prevention stresses thorough removal from all tooth surfaces of the adherent microbial masses. Most suggest brush, floss and rinsing as essential parts of the daily routine of mouth care.

The essential factor in the program for

**Basic Fuchsin Disclosing Stain for Demonstrating Mouth Organisms: Dissolve 6 gm. basic fuchsin in 100 c.c. of 95% ethyl alcohol to make a saturated stock solution. Bottle in ½ oz. bottles with dropper corks. Twelve drops of the stock solution are added to 2 tbs. of water in a paper cup. The patient is requested to rinse thoroughly with the solution for one-half minute and to spit it back in the cup. It is then poured down the drain and the patient rinses with clear water until excess stain is removed. Basic fuchsin is water soluble, is easily washed off most things but will stain rayon. It fades from teeth and organisms in about 4 hours when not removed by patient or dentist. It may be obtained from Allied Chemical & Dye Corp., National Aniline Division, 40 Rector Street, New York, N. Y.

prevention is found in the education of the patient by the dentist.[4] The patient must know periodontal disease is caused by the periodontal microcosm. The dentist must perform all necessary periodontal treatments with strict, systematic and complete application. At each appointment the patient's condition is checked with the aid of disclosing solution. The patients' responsibility for the success of prevention of periodontal disease must be stressed. It is this combination of intelligent understanding and clinical application of basic knowledge concerning the etiology and pathogenesis of periodontal disease which leads to successful treatment and prevention.

L. S. Parmly[24] wrote in a course of lectures on this and other dental subjects in 1821, "The commencement of disease is too generally looked upon as a matter of little importance, and thus but few persons take any notice of its progress until the agonizing pain of toothache, or deformity, forces it upon their consideration; . . . Hence the most useful advice with which I can conclude these lectures is by urging on every individual the necessity of aiming at the prevention of disease altogether, which can in a great measure be effected without engrossing more time than its importance merits."

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Periodontal Traumatism

by OSCAR BJORNDAHL, D.D.S., *St. Paul, Minnesota*

IN LIGHT of the controversy that existed only a few years ago, regarding the relative importance of traumatic occlusion as an etiological factor in periodontal disease, it is of mentionable interest that today some of the classifications list Periodontal Traumatism as a separate entity. It must be with an accomplished satisfaction and pride, to those men who for years have alleged trauma as being an important clinical factor, to have traumatic occlusion recognized, classified, and named Periodontal Traumatism. Notably among these men are Box, Stillman, McCall, Miller, and Sorrin. Only recently Glickman[1] stated that there seems to be a significant relationship between trauma and the formation of the intra-bony or vertical type of pocket.

The selective grinding of tooth surfaces to equilibrate the forces of stress upon the teeth, is today an accepted and necessary procedure for many of our periodontal cases. The correction of occlusal disharmonies can be as challenging and stimulating as any other phase of dental practice. In fact, there is no phase of dentistry that is of as great importance to good dentistry as a balanced occlusion. Whether it pertains to the adjusting of occlusal disharmonies, the construction of a bridge or denture, or to a filling in the individual tooth. Orban[2] wrote regarding balanced occlusion: "The harmonious relation between teeth is the basic objective of a biologic functional approach to all dentistry."

The classification for periodontal disease as used at the Michigan Workshop[3] had the four general headings of Gingivitis, Periodontitis, Periodontosis, and Periodontal Traumatism. The most common clinical and radiographic symptoms of traumatism are:

Clinical:

1. Loosening of teeth.

2. Patient complains of soreness when teeth are brought in contact or when chewing.
3. Sensitivity to percussion, sometimes cold.
4. Premature contacts.
5. Interfering cusps.

Radiographic:

1. Widening of periodontal space.
2. Uneven thickening of lamina dura.
3. Bone resorption.
4. Sometimes root resorption.

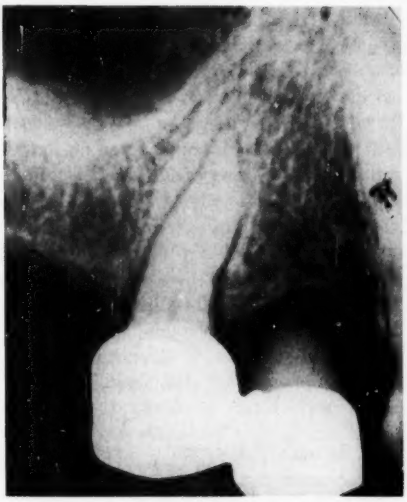
Knowing the clinical and roentgenographic symptoms, some consideration to the factors relative to this problem of periodontal traumatism is in order. To better understand periodontal traumatism or traumatic occlusion, one might quickly mention the other types of occlusion found in the mouth. The terms normal occlusion and malocclusion are familiar to all. The mechanical or geometric aspects of the occlusion, that is the relation of one opposing tooth to the other, or of the upper and lower arches to each other is important as well as the interdigitation of the cusps, the steepness of the incline planes, their relation to each other in the various positions and excursions, and the amount of overjet and overbite. If all these factors more or less follow the pattern associated with the setting up of a full upper and lower denture, the occlusion can be accepted as normal. Any deviation from this normal is spoken of as malocclusion.

Problems of the periodontist are biological rather than mechanical. Occlusal problems as they relate to the disease periodontal traumatism, then, are either physiological or pathological and not normal or malocclusion. Therefore, normal occlusion becomes physiological occlusion, and any deviation from this becomes pathological or most often referred to as traumatic, rather than malocclusion. Physiology, being the science dealing with the normal function of living tissues, the term physiological occlusion, then can only concern itself with the response of the living tissue to an occlusal factor.

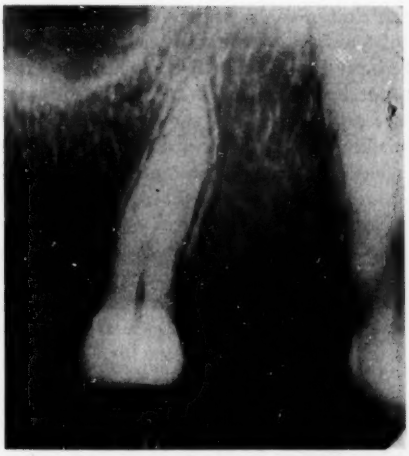
It is known that teeth are suspended or cushioned in living tissue, namely the cementum, periodontal membrane, and bone. During mastication, or the act of occluding the teeth, a certain amount of force is required to produce these actions or movements. These forces produce a stress or strain upon the supporting tissues and these tissues must offer resistance to these forces. The size or thickness of these supporting tissues, mainly the periodontal membrane, changes according to the functional demand. These various sizes of the periodontal membrane are named the biologic, physiologic, and pathologic widths.[4] The biologic width is very thin and is found in nonfunctioning teeth; the physiologic width, somewhat thicker, is found in teeth in normal function; and the pathologic width refers to a thicker and uneven width of the membrane associated with Periodontal Traumatism. (Roentgenograms A, B and C.)

With this knowledge, one may now attempt to define physiological occlusion. Knowing that the teeth are subjected to constant occluding forces, and that these forces are sustained by the supporting tissues, physiological occlusion can be defined as the state of equilibrium that exists between the forces acting upon the teeth during contact and the resistance of the

Roentgenograph A—Shows a typical radiographic picture of periodontal traumatism. Note the mesial root of first molar, with its widened periodontal space and uneven thickening of lamina dura. Also note formation of intrabony type of pocket mesially of same tooth.



Roentgenograph B—Note uniform thickening of entire periodontal space, also increase in size and density of lamina dura. Etiology—Faulty restorative work—wing type bridge, causing excessive function. Treatment—removal of bridge.



Roentgenograph C—Same as B. Patient returned after two years absence. Note complete repair of supporting structures, the more normal width of periodontal space and lamina dura.

supporting tissues to these forces. To help in remembering the definition, the following equation can be formulated.

Physiological occlusion exists when occlusal force = Resistance of supporting tissue

As long as these two factors of force and resistance balance each other, physiological occlusion exists entirely independent of the geometric or mechanical relation of one tooth to the other. In other words, do not confuse malocclusion as meaning traumatic occlusion. Much too often is the malrelation of one tooth, or the teeth, to each other referred to as being traumatic occlusion. However, clinical and radiographic examination reveal no traumatic symptoms and, therefore, from a periodontal standpoint, must be regarded in physiologic relation. Malocclusion does not mean traumatic occlusion, but in many instances can be the etiological factor for the trauma that exists.

Clinical application of this knowledge is important. Mouths are seen badly mutilated from malposition, missing teeth, or ill fitting restorations, so that the entire force of occlusion is carried by only a few teeth, and still no symptoms of traumatism exist. The only possible explanation for these particular cases is that the tissues of these patients have an adaptability or tolerance to these forces that do not follow the usual pattern of force and tissue response. Clinicians can not definitely predict trouble for every patient with malrelated teeth. It is known from experience and general observation, that it is not necessary to correct every case of malrelation. In other words, if changes are made in the occlusal patterns, one must first have symptoms or reasons to justify the procedure. A recent article by Reeves,[5] "Is Prophylactic Occlusal Adjustment a Justifiable Procedure?" confirms the need for caution of the indiscriminate grinding of teeth. However, it is good professional judgment to acquaint patients with the possibilities that exist. Particularly is this important with the younger age group. A study of the case, as to the age of the patient, the number of years malrelation has existed, and the recognition of traumatic symptoms, will guide in planning treatment.

Miller[6] defines traumatic occlusion, "as

an abnormal occlusal stress that is capable of producing or has produced injury to the periodontal tissue." However, the periodontal tissues like all other living tissues, are capable of repair and rebuilding. These tissues must adapt themselves to the continually changing functional demand required of our teeth. Therefore, new cementum, new periodontal fibers, and new bone are constantly being formed to maintain this physiologic balance, so necessary for survival of any living tissue. Clinically, it is well known that soreness accompanies a tooth when a filling is too high and relief follows when the high spot is relieved. The soreness produced was due to the temporary interference of the balance that exists between function and tissue response. When studying periodontal traumatism, a disturbed relationship between the factors of occlusal stress and the factors of resisting periodontal stress must be considered. Therefore, one of these factors is abnormal in scope. This is the basis for Box's[7a] Classification of Traumatic occlusion, which he lists as following:

1. Primary Factors
   Abnormal occlusal factor with normal periodontal factor. The occlusal factor is such that over-stress is produced in the periodontal tissues.
   a. Cusp Interference—
      Malocclusion. Faulty dental restorations. Unbalanced orthodontia cases.
   b. Uneven Wear—
      Production of facets or concavities tends to greater torque, force for mastication is greatly increased.
   c. Faulty Restorations—
      Tend to increase torque. Poor anatomical reproduction. Bulky restoration. Clasp or attachments for partial dentures.
   d. Dental Caries—
      Loss of dental anatomy. Food Impactions.
   e. Excessive Wear—
      Loss of cusps and sulci, produce heavy torque.
   f. Lack of Occlusal Vents—
      Increases stress, for sluiceways are absent, requires heavier mastication.
   g. Loss of Teeth—
      Causing shifting, force no longer in direction of long axis of tooth.
   h. Habits—
      Chewing of extremely hard foods. Pipe smoking, cigarette holders, etc.
      Occupational—Biting thread. Holding nails, etc.
      Nervous —Bruxism. Biting fingernails, lips, etc.
2. Secondary Factors
   Normal occlusal factor with abnormal periodontal factor. The magnitude of the occlusal force may be normal, but due to loss of supporting tissue over-stress is produced.
   a. Periodontitis.
   b. Periodontosis.
3. Combined Factors
   Both occlusal and periodontal factors are abnormal. Any of the primary factors, with loss of supporting tissues, unless the primary factors can be corrected, tooth is soon lost.

From the classification presented above will be noted that periodontal traumatism is characterized by alterations of the supporting tissue and looseness of the teeth. However, again, one must not be unmindful of the fact that each tooth has a certain amount of mobility and this movement is spoken of as its functional or physiologic limit. Any movement beyond this limit causes damage to the supporting tissues and therefore becomes traumatic. The teeth are subjected to two types of forces or strains, namely vertical and horizontal, often referred to as axial and lateral. Box[7b] refers to these excessive forces as vertical and horizontal overloading; both are descriptive and applicable to the problem of periodontal traumatism.

A tooth, because of its root form (coni-

cal in shape), supporting bony architecture (the more dense cortical layer of bone), and the general arrangement of periodontal fibers (mainly the oblique fibers which extend from the cementum in an upward direction to the alveolar process), is well fortified to withstand vertical overloading. This is explained by the fact that as a tooth is depressed into its socket, this vertical force is sustained by the resistance force as a whole. These resistance structures (cementum, periodontal fibers, and bone) respond as a unit, and thereby, the force or injury is greatly minimized. Teeth can withstand many pounds of pressure in the direction of the long axis of the tooth, but only a fraction of this in the lateral direction. This fact is substantiated clinically in that seldom with finger pressure can a tooth be depressed into its socket. On rare occasions we may find a tooth that can be depressed, but such a tooth always has an unfavorable or poor prognosis.

The tissues' changes that occur when a horizontal overloading exists has for years been a favorite study among many investigators. There are many reports of both animal and human studies, and the findings have been more or less the same. There is a general agreement that as the tooth is being moved or tipped horizontally, that the fulcrum point is somewhere between the middle of the root and the apex. This rotating or pivoting of the tooth from the fulcrum point prevents the supporting structures from responding as a unit, as in the case of a vertical stress. Therefore, areas of unequal stress, known as the pressure and tension areas are present. These various studies have found that the stretching of the fibers on the tension side produces a stimulating response for the laying down of new bone. Whereas, the opposite occurs in the pressure areas, that is the stimulus causes bone resorption. Now as long as these forces stay within the functional limits of tooth mobility, repair and rebuilding take place and no particular damage results. However, when these forces are so excessive and prolonged that the periodontal membrane can no longer maintain its physiologic relation, a severe and pathologic change takes place in the supporting structures. Particularly is this true on the pressure side, because the periodontal membrane is crushed between the root and bone. This crushing of the membrane causes not only a necrosis of the membrane, but also a resorption of the bone in these pressure areas. The tooth becomes loose and sore, so that now we have the symptoms of periodontal traumatism both clinically and radiographically.

From this brief discussion, it should now be understandable that occlusal trauma is capable of destroying this biologic balance between force and resistance so necessary for tissue health. Nor should it be too difficult to assume, that this tissue injury is due to trauma, the primary factor for the periodontal condition known as periodontal traumatism. If an inflammatory condition already exists in the gingiva, it should not be hard to believe that this inflammation could rapidly extend into these traumatized areas and produce a severe periodontal condition. That occlusal trauma is a primary etiological factor for periodontal traumatism and that it also is a predisposing factor for a more severe periodontal condition, is only restating what Orban[2] said in one of his recent papers.

Clinical application of this knowledge and treatment will be to correct these occlusal disharmonies from a traumatic to a physiologic relation. In other words, and in most instances, it will be changing the occlusion from the more damaging horizontal force to the more favorable vertical. It is most satisfying to have these loose and often sore teeth to again become firm and comfortable.

There are some rather definite principles and rules to follow for correcting occlusal disharmonies. These rules, if understood and followed, avoid doing irreparable damage. Also they offer a more or less systematic procedure to follow. The term selective grinding is often used by some men, for it denotes a word of caution, that grinding the teeth must be done with an under-

standing of what one wishes to accomplish. Many[4b, 6b, 8, 9] of the text books have excellent chapters on the methods to follow when grinding the teeth. There are also a number[2, 10, 11] of papers on this subject. A careful study of this material makes selective grinding possible for any interested dentist. To simply read and attempt to visualize what the author is saying, is often very confusing and discouraging. Study models mounted on an articulator to demonstrate to oneself each step as given, when studying what the author is saying, is the most helpful and quickest manner to learn their rules and principles.

If periodontists will acquaint themselves with the available knowledge on this subject of periodontal traumatism, the problem of correcting occlusal disharmonies can be made with a more understandable approach.

OUTLINE OF RULES FOR SELECTIVE GRINDING

Definitions:

1. *Centric Relation* is defined as that maxillomandibular relationship when the condyles are in their most retruded position in the glenoid fossae from which any eccentric movement can be made.
2. *Hinge Position:* (Orban) The most retruded position of the mandible which the patient can voluntarily achieve with own musculature. This position coincides with apex of gothic arch tracing.
3. *Centric Occlusion:* True centric occlusion exists when the intercuspation of the teeth is in harmony with mandibular hinge position and the muscles of mastication.
4. *Habitual Centric:* An acquired or convenience position determined by the intercuspation of the teeth, usually forward of the hinge or true centric position.

General Rules:

1. *Before grinding:* Make study of case, note contact relation of opposing teeth in all positions and excursions. Study casts are most helpful.
2. Avoid grinding cusp tips as much as possible, they are needed for balancing the eccentric positions.
3. Never grind or take teeth out of centric contact.

Objectives:—(Orban)

1. Grind so that occlusal forces are directed to the long axis of teeth. Lingual cusps of upper into the central grooves of lower teeth. Buccal cusps of lower into the central fossae of upper.
2. Distribute forces in functional position to largest possible number of teeth.
3. Eliminate premature contacts on balancing side.
4. Establish static relation between cusps and opposing fossae.
5. Reduce steepness of cusp inclines. Establish similar inclines on all teeth.
6. Reduce size of contact surfaces.
7. Retain sharpness of cusps and establish smooth guiding planes.

Three Occlusal Relations

I. Hinge or Centric Position.

II. Protrusive Excursion and Position.

III. Lateral Excursion and Position.

Important: Before grinding locate corresponding mark on opposing tooth, if not certain, using different colored carbon paper, super-imposed on mark in question, is most helpful to verify correct place to grind.

When grinding work with one relation or position at a time, that is when working with centric, do not have patient make lateral movements.

RULES FOR GRINDING CENTRIC RELATION

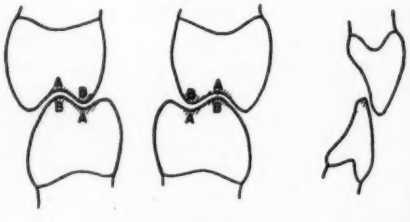


Fig. 1

I. Grind Fossae (A)
   1. If cusp has premature contact in centric, but is in harmony in eccentric positions.
   2. If there is no contact in eccentric positions, grinding of cusp would only increase space.

II. Grind Cusp (B)
   If it contacts prematurely in all eccentric positions.

III. Grind Incisal Edges Lower Incisors
   Grinding uppers would steepen lingual incline, avoid taking out of centric if possible.

RULES FOR GRINDING HINGE RELATION

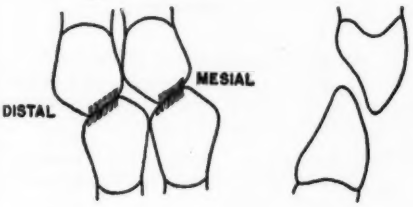


Fig. 2

I. Grind—Rule M.U.D.L. (Mesial Upper, Distal Lower)

When mandible must be re-positioned in a retruded position from habitual centric.

1. Mesial Incline Upper—Usually lingual cusp. Tip of Cusp is moved distally.
2. Distal Incline Lower—Usually Buccal cusp.

Tips of cusps are moved mesially.

Important: After centric balance has been obtained, confine grinding to incline planes and away from point of centric contact; do not include point of contact. It is well to use one color for centric, another for all other relations.

RULES FOR GRINDING PROTRUSIVE EXCURSION AND POSITION

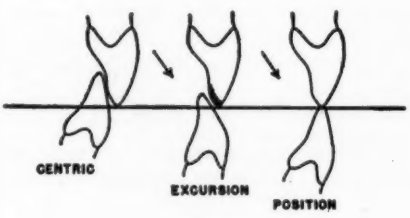


Fig. 3

RULES FOR EXCURSION

Forward Movement of mandible from centric

I. Grind Posteriors: Look for marking primarily on distal lingual inclines of buccal upper and mesial buccal inclines of lingual lowers.

II. Grind Anteriors: Lingual inclines of upper anteriors.

1. Do not grind point of centric contact.
2. Grinding lowers will produce loss of centric contact.

RULES FOR POSITION

End to end relation of anterior teeth

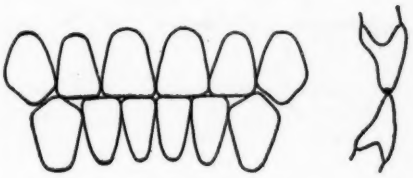


Fig. 4

I. Grind Uppers: To produce equal pressure on as many teeth as possible.

1. Esthetics often governs amount of grinding.
2. Grinding lowers will produce loss of centric contact.
3. If little more grinding will produce posterior contact, do so.
4. If deep overbite, lowers can often be shortened without loss of centric contact.

RULES FOR GRINDING LATERAL EXCURSION AND POSITION

Working and Balancing Side—Right or Left
Rule "BULL"—Buccal uppers, Lingual lowers

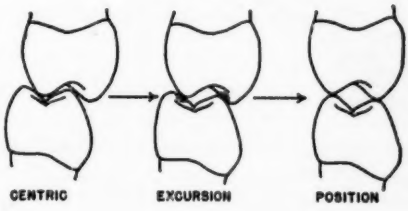


Fig. 5

RULES FOR EXCURSION

Incline Planes

I. Working Side—Grind inner incline planes—Buccal upper, lingual lower.

1. Grinding of lower buccal cusp or lingual upper will interfere with centric.
2. Always grind from point of centric, do not include it.
3. If *cuspid* teeth interfere, grind upper lingual from point of centric contact along path to incisal edge, grinding lower destroys centric contact.

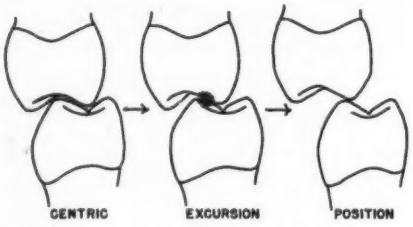


Fig. 6

II. Balancing Side—Grind inner incline planes—Buccal incline upper lingual cusp. Lingual incline lower buccal cusp.

THREE COMMONLY USED METHODS FOR OBTAINING HINGE OR TRUE CENTRIC RELATION

1. Relaxed opening and closing motions by the patient, guided by operator's hand into the most retruded position. Wax registration for cast mountings. (Lauritzen.)
2. Locating rest position through proper patient training and plaster registrations. (Ralph Boos.)
3. Use of clutch and gothic arch tracing. (Lichtman.)

RULES FOR LATERAL POSITION

Cusp to Cusp Relation

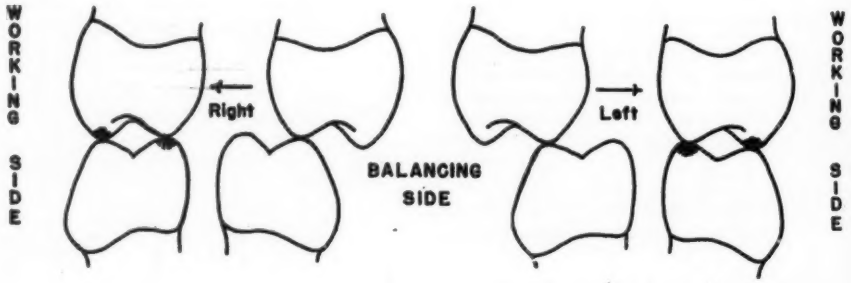


Fig. 7

I. Working side—Grind upper buccal cusps, lower lingual cusp. Grinding these cusps cannot interfere with centric.

II. Balancing Side—Grind with care, as here we must grind the cusps that maintain centric (upper lingual, lower buccal). Decide which cusp is most helpful in maintaining centric, then grind the other.

*

CUSP AND INCLINE PLANE RELATION

In the above "Rules for Grinding" the word "Bull" is used in several places. That there might be those not familiar with the term Bull, a short explanation seems desirable. The four letters of the word "BULL" designate the Buccal Upper, Lower Lingual cusps of the posterior teeth. This is a rather common term used by many when teaching the rules for selective grinding. The rule "Bull" applies when grinding the posterior teeth in lateral excursion and position. The *lower* buccal and *upper* lingual cusps are the "centric maintaining" cusps, therefore, the term "Bull" is a simple means in helping to remember that one can grind the upper buccal and lower lingual cuspal areas with little danger of disturbing centric. In other words, "Bull" helps in selecting the areas to be ground with some degree of safety without doing too much damage.

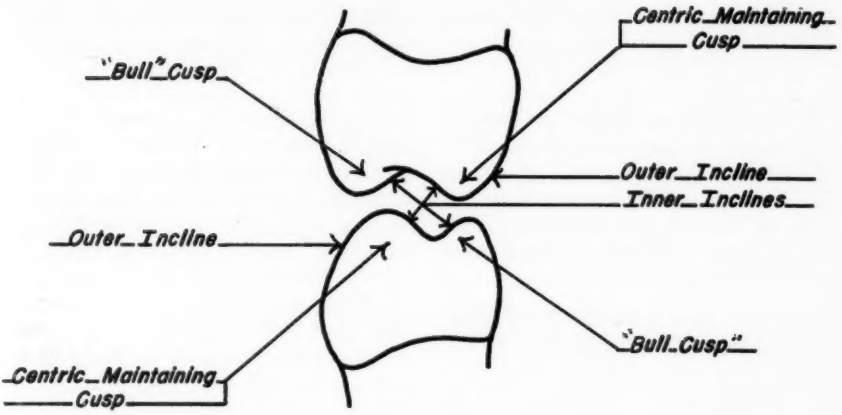


Fig. 8

Centric Cusps are Upper Lingual and Lower Buccal. BULL Cusps are Upper Buccal and Lower Lingual.

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FEDERACION ODONTOLOGICA CENTRO AMERICA-PANAMA

The Second International Convention of the Odontological Federation of Central América-Panamá (Federación Odontológica de Centro América-Panamá) (FOCAP), will be held in San José, Costa Rica, in the University of Costa Rica building of Basic Sciences, from November 25 to November 29, 1958.

The subscription fee will be the amount of $20 U. S. and will enable one to participate in both the scientific and social events of the meeting.

Anyone who may wish to participate in the scientific section of the meeting, please write Raymond Pauly, S.D.D.S., Associate Secretary, Apartado Postal 698, San Jose, Costa Rica, C. A.

What is Known in Periodontology

by FRANK M. WENTZ, D.D.S., M.S., PH.D., *Chicago, Illinois*

THE aim of periodontal treatment is to keep the supporting tissues of the teeth in optimal physiologic condition. In order to understand the physiology and pathology of the supporting tissues, it is necessary to look upon them as tissues in a state of flux.

The ever-constant changes in cementum, bone and periodontal membrane are directly correlated to attrition which, in turn, is the inevitable result of function in the normal masticatory organ. For instance, occlusal and incisal wear are compensated by the continual vertical eruption of the teeth so that the occlusal facial height remains constant, allowing optimal function of the masticatory muscles. However, in analyzing the effects of wear, it must not be overlooked that attrition is not only occlusal and incisal but also proximal wear. The loss of mesio-distal diameter of the teeth is compensated for by mesial drift, allowing the teeth to always keep sharp proximal contact.

The complicated movements of the teeth compensating for physiologic wear are brought about by differential growth of alveolar bone and cementum, which are processes that necessitate a continual rearrangement of the principal fibers of the periodontal membrane.

This necessary state of flux of the supporting tissues to maintain an ever-constant adjustment of the teeth for optimal function is best maintained if it is not compromised by disease or occlusal disharmonies. While the capacity of the supporting tissues to adapt is very great under optimal health conditions and in the young, it is decreased and may not be adequate under less favorable health, aging, dietary, nutritional or systemic influences, and should therefore be treated.

One factor, then, in an integrated program of periodontal therapy, in addition to treating diseases that threaten the supporting tissues, is to maintain or restore an optimal functional occlusal relationship between opposing arches. Malocclusions, occlusal disharmonies, cusp interferences, premature contacts, missing teeth, irregular migration and tipping, unphysiologic dental restorations, all disturb the ideal functional relation of the teeth. When the supporting tissues of the dentition are diseased, when natural functional adaptation of the tissues are impaired, it is of paramount importance to have the most ideal occlusal relation to prevent the secondary damage inflicted by occlusal disharmonies.

Any local treatment will be effective only if the supporting tissues of the teeth are regarded and understood as part of the masticatory organ. Therefore, the broadest possible approach to the problem of occlusal equilibration is the only promising approach. In the analysis of a case, the dynamic and functional visualization of the dentition is of primary necessity. Here, of course, the occlusion of the teeth and their articulation has to be examined and this in turn involves an understanding of the temporomandibular joint, its musculature and its movements.

THE FUNCTIONAL UNIT

It has often been stated that the masticatory organ consists primarily of the teeth and its supporting tissues, the musculature and the temporomandibular joint, though cheeks and tongue also could be included. Though valid, the idea of a masticatory organ as a unit is too general to convey the significance of the interdependence of its parts. It is necessary to break this statement down to concrete examples of these interrelations to convey the concept that the single structures of the masticatory organ are not independent, not dependent, but interdependent.

The following examples will illustrate. The teeth while they erupt attain their definitive position under the influence of muscle forces. Once the teeth are erupted and in position, they in turn influence the masticatory movements of the mandible and thus exert their influence upon the muscles. The periodontal membranes are generally considered merely as suspensory ligaments of the tooth. As such, the arrangement of the principal fibers is dependent upon the forces exerted during function; that is, muscle action influences the structure of the periodontal membrane. It must, however, be stressed that the periodontal membrane is a most important sensory organ richly supplied with proprioceptive nerves and their endings. These nerve endings when stimulated by stretching or releasing of the principal fibers, are the most important sources of reflectory control of the mandibular musculature.

This musculature exerts its influence upon the temporomandibular joint not only by moving the disk and mandible but also by maintaining the balance of the different constituent parts of the joint. In turn, just as in other parts of the body, the sensory nerve endings in the capsule and ligaments of the joint influence its musculature and are indispensable for proper co-ordination of the neuro-musculature apparatus.

It is also clear that the periodontal membranes of the teeth and the guide planes on cusps and incisors influence not only the musculature but also the temporomandibular joint, just as the free normal mobility of the temporomandibular joint is indispensable for the proper attrition pattern of the dentition.

In other words, no part of the masticatory organ is either independent of the other or exerts any influence without in turn being influenced by all the other parts. If this is true during normal function of a healthy masticatory system, it must also be true, that any disturbance of any part of this system will be injurious to any other part. Thus, it may be said that a misplaced filling, for example, in one tooth may well spell the ruin of the harmony of the entire masticatory apparatus. The unphysiologic inclination of a cuspal plane may lead to unphysiologic stresses in the periodontal membrane of this tooth and possible even of neighboring teeth and their antagonists. Then unphysiologic impulses of proprioceptive nature are fed back to the mandibular musculature via reflex arcs, destroying the delicately balanced harmony of correlated muscle action. The irritation of the musculature leads to spastic contractions or to what may be called "panic" or "confusion" of the musculature and then the proper balance in the temporomandibular joint is endangered and finally lost. Degenerative processes in the joint follow and then all the teeth are stressed in an unphysiologic way qualitatively as well as quantitatively, and the result may be traumatic changes in the supporting tissues of these teeth, acting as a serious aggravating factor of any periodontal disease present leading to the destruction of the supporting mechanism of the teeth.

Two key concepts therefore form the basis of "What is established in periodontology."

1. The biologic concept.

2. The concept of inter dependence of the parts of the functional unit—the masticatory organ.

This background will be the frame to answer the questions of what we know about gingivitis and periodontitis.

I. What is known about the etiology of periodontitis?

Periodontitis is primarily due to local irritation and bacterial action. Occlusal traumatism is not a cause, but may be a serious aggravating factor.

The initial lesion is a gingivitis. The chronic inflammation with its production of toxic products leads to destruction of

the epithelial attachment and connective tissue fibers. Deepening of the pockets and alveolar crest resorption follows. There is no systemic disease or systemic condition which in itself would produce a periodontitis. However systemic conditions are serious aggravating and modifying factors of the initial periodontal lesion.

Why is the periodontium more vulnerable? This is because the periodontium is in a constant state of growth and adaptation to function (flux) as previously emphasized in the initial concept.

Occlusal traumatism with its added damage upon compromised tissues will cause further aggravation of the condition.

The primary etiology of periodontitis is local irritation due in the main to neglected oral hygiene.

II. Is periodontitis curable?

The answer is yes, but what is inferred by cure must be defined. A cure means the absence of a disease process, though impairment may be present. A comparison may bring home this point. All diseases may be classified in two groups. In one group are such diseases as measles or appendicitis or dental caries; when treated satisfactorily, no impairment remains and the patient does not have to see physician, surgeon or dentist again for the treatment of his past ailment. In another group are diseases like tuberculosis or periodontitis. They also can be cured; however, a certain impairment due f.i. to some loss of bone support or to the original contributory systemic factors may remain. For this reason periodontal patients must submit to regular careful periodontal check-up the same as patients with arrested tuberculosis.

We cannot expect regeneration of lost periodontium. Therefore we suffer a functional impairment in this extent. The degree of impairment depends upon how far the disease had advanced when therapy was instituted. The more the tissue loss the more difficult the functional problem. Here the understanding and correction of occlusal traumatism plays its greatest role. The dentition may be lost, if occlusal interferences which aggravate and further damage the impaired condition of the periodontium are not eliminated.

Periodontitis is *curable* if the inciting etiologic factors (generally dental deposits) are completely eliminated and the functional impairment compensated for and controlled.

III. Are the available periodontal treatment methods sufficiently standardized to be applicable for the majority of cases of periodontitis?

The answer is yes, the treatment methods are standardized.

The aim of therapy is to keep the supporting tissues in an optimal physiologic and hygienic condition, which can be maintained!

The treatment is not the problem. Maintenance of the cure is the problem.

The logical regimen of procedure follows:

1. Proper diagnosis.
2. Proper prognosis—calls upon the sum total of your experience.
3. Proper treatment plan.

If the etiology of periodontitis is known —the therapy is to remove it! The dental deposits comprise this etiology. The handling of the impairment of the condition is based upon the logical knowledge of the function of the masticatory organ as a unit. We treat this.

However, there is a truism. We don't treat cases, we treat individuals. Individual patients vary. The skill of the clinician will spell success or failure here.

IV. Is periodontitis preventable?

The answer is yes! We know the etiology. This is local irritation caused by:

1. Dental deposits.
2. Poor dentistry.

One of the important reasons for neglect of the early periodontal lesions is the fact that they seldom cause pain or discomfort; the patient remains, therefore, often unaware of the insidiously progressing disease. The dentists, on the other hand, must train themselves to recognize the early signs of periodontal disease. They must pay attention in minute detail to changes in the color, texture and contour of the gingiva. They must scrutinize carefully the patient's roentgenograms which would be of the highest quality and must be retaken at least every two years. Particularly must they scrutinize these roentgenograms for the presence of spurs of subgingival calculus and fillings with overhanging gingival margins, rough or ill-shaped contacts or imperfectly formed embrasure space.

CONCLUSION

A firm clear basis of understanding of the normal structural relations of the periodontium is a pre-requisite for successful periodontal therapy. In this light, further understanding of the biology and physiology of the attachment apparatus of the teeth will guide us to better and more precise methods of therapy.

THE AMERICAN INSTITUTE OF DENTAL MEDICINE

The 15th Anniversary Meeting of The American Institute of Dental Medicine will be held in Palm Springs, California, October 12-16, 1958.

Dr. E. Cheraskin, Professor of Oral Surgery and Oral Medicine, School of Dentistry, University of Alabama, will speak on selected subjects from the field of oral Biology. Dr. S. I. Hayakawa, San Francisco State College, will discuss "communication" and related subjects in general semantics. Dr. Joseph C. Muhler, Associate Professor in Biochemistry, Nutrition, and Preventive Dentistry at Indiana University, will lecture on the relationship of the endocrine system to dental caries, and fluorine metabolism. Dr. Hans Selye, University of Montreal, will present his world-renowned concept of "stress," and the "focal syndrome." Dr. H. M. Worth, from Victoria, British Columbia, will describe the roentgenographic aspects of benign and malignant tumors of the jaws and their differential diagnosis.

Dr. Herbert M. Evans and Dr. K. F. Meyer, authorities in the fields of hormones of the pituitary gland, and virus diseases, respectively, will be featured speakers. The morning sessions will be devoted to presentations of the above subject matters, to be followed in the afternoons by small, informal seminars, optional to the participants. The entire Thursday morning will be utilized in an Open Forum to apply the most important aspects presented to problems in dental practice. Doctor H. Becks will serve as Moderator.

"Diagnostic Clinics" will be introduced this year as a special feature, and the participants will be encouraged to bring patient problems with pertinent medical aspects, diagnostic data, roentgenograms, etc., for consultation and clarification. In this way, it will be possible for the members to avail themselves of the knowledge and experience of the experts present among the participants and lecturers representing nearly every discipline in Dentistry.

The official program, with full information, may be secured from the Executive Secretary, Miss M. Lewis, 2240 Channing Way, Berkeley 4, California.

PERIODONTOLOGY WORKSHOP SUCCESSFUL

The Workshop for Teachers in Periodontology sponsored by the American Academy of Periodontology was held at the University of California, School of Dentistry, San Francisco, June 23rd, 24th & 25th, 1958. It was in all respects a most successful conference in which were represented all of the dental schools in the United States and Canada with the exception of one. The participants, numbering 92 in all, met in respective committees and deliberated upon the subjects and charges previously prepared for each. All of the participants were active teachers in Periodontology who had been nominated for attendance by their respective deans. This brought together a cohesive body which applied itself to the problems at hand and the improvement of the teaching of Periodontology within the dental curricula of the represented universities. The work of the committees was prosecuted zealously and solutions to most of the problems were proposed. Each committee chairman read before the entire body, on the final day, a report representing the conclusions of his specific committee. The body then approved the respective reports with certain amendments, corrections and deletions. These reports with the accompanying story of the Workshop and its suggestions in the direction of better teaching of Periodontology will be published by means of a grant from the National Institute for Dental Research. Copies of this publication will be sent to all universities, to the members of the American Academy of Periodontology and to other interested institutions as well as individuals. Listed below are the subjects of the various committees and the names of the respective chairmen, consultants, secretaries and participants:

COMMITTEE NO. I

The Techniques of Teaching in Periodontology. Academic Student Records & Audio Visual Aids.

Consultant: Dr. Harold Ray

Chairman: Dr. Frank E. Beube, Columbia Univ. 630 W. 168th St., N. Y. 32, N. Y.
Secretary: Dr. Gerald Mitchell, Coll. of Medical Evangelists, Loma Linda, Calif.

Members

Dr. L. C. Alexander, Marquette University, Milwaukee 3, Wisconsin
Dr. E. L. Banks, Emory Univ., School of Dentistry, Atlanta 3, Ga.
Dr. S. O. Banks, Meharry Medical Coll., Sc. of Dentistry, Nashville 8, Tenn.
Dr. Martin Cattoni, Univ. of Texas, Houston 4, Texas.
Dr. Abram Chasens, Fairleigh-Dickinson Univ., Teaneck, N. J.
Dr. Robert Keeffe, Univ. of Calif., U. C. Medical Center, S. F. 22, Calif.
Dr. D. Gordon Pentz, Dalhousie Univ., Halifax, N. S.
Dr. Harold Santos, Univ. of Calif., U. C. Medical Center, S. F. 22, Calif.
Dr. Walter C. Stout, Baylor Univ., Dallas 10, Texas.
Dr. George J. Witkin, New York Univ., N. Y. 10, N. Y.
Dr. W. F. Walford, 1414 Drummond St., Montreal, Canada.
Dr. Vernon E. Hyde, 940 North Van Ness Ave., Fresno, Calif.

COMMITTEE NO. II

Didactic Subject Matter Prequisite to and of a Major Concern Within the Responsibility of Periodontology. Integration of Basic Sciences.

Consultant: Dr. Maynard K. Hine

Chairman: Dr. Donald Kerr, Univ. of Michigan, Sc. of Dentistry, Ann Arbor, Mich.
Secretary: Dr. C. Kenneth Collings, Baylor U., Sc. of Dent., Dallas 10, Texas.

Members

Dr. Edward F. Allen, Washington Univ., Sc. of Dent., St. Louis 10, Mo.

Dr. D. Walter Cohen, U. of Pennsylvania, Sc. of Dent., Phila. 4, Pa.
Dr. Glen Curtis, Coll. of Medical Evangelists, Loma Linda, Calif.
Dr. Benjamin Dienstein, U. of Calif., U. C. Medical Center, S. F. 22, Calif.
Dr. Anthony Garguilo, Loyola Univ., Coll. Dental Surgery, Chicago 12, Ill.
Dr. Paul Goldhaber, Harvard Sc. of Dental Med., 25 Shattuck St., Boston, Mass.
Dr. Thos. Holder, Univ. of Oregon, Dental School, Portland 1, Oregon.
Dr. Marjorie Houston, Northwestern U., The Dental School, Chicago 11, Ill.
Dr. Grover C. Hunter, Univ. of N. Carolina, Chapel Hill, No. Carolina.
Dr. Saul Schluger, Univ. of Washington, Sc. of Dent., Seattle 5, Wash.
Dr. Wm. R. Wolfe, Univ. of Louisville, Sc. of Dent., Louisville 2, Ky.
Dr. T. E. Bolden, Seton Hall Coll. of Medicine & Dent., Jersey City 4, N. J.
Dr. Nathan Friedman, Univ. of So. Calif., Sc. of Dent., 925 W. 34th St., L. A. 7.

COMMITTEE NO. III

Subject Matter and Scope of Undergraduate Clinical Periodontics. Postgraduate and Graduate Projection.

Consultant: Dr. Balint Orban

Chairman: Dr. B. O. A. Thomas, 668 Homer Ave., Palo Alto, Calif.
Secretary: Dr. John R. Wilson, Ohio State Univ., Coll. of Dent., Columbus 10, Ohio.

Members

Dr. W. Robt. Piddington, Univ. of Maryland, Baltimore 1, Maryland.
Dr. Robt. J. Bruckner, Western Reserve Univ., Cleveland 6, Ohio.
Dr. W. Hinson Jones, Univ. of Buffalo, Sc. of Dent., Buffalo 16, N. Y.
Dr. Ben Klavan, Univ. of Illinois, Coll. of Dent., Chicago 12, Ill.
Dr. Geo. C. Lowther, Univ. of Minnesota, Minneapolis 14, Minn.
Dr. Nicholas Marfino, Loyola Univ., Sc. of Dent., Chicago 12, Ill.
Dr. Robert Oslar, Coll. of Physicians & Surgeons, 344-14th St., S. F., Calif.
Dr. R. Earl Robinson, Univ. of Calif., U. C. Medical Center, S. F. 22, Calif.
Dr. Perry Ratcliff, Univ. of So. Calif., 925 W. 34th St., Los Angeles, 7, Calif.
Dr. H. L. Samuels, Univ. of Alberta, Edmonton, Canada.
Dr. Geo. M. Stewart, Univ. of Pittsburgh, Pittsburgh 13, Penna.
Dr. Henry Swenson, Univ. of Indiana, 1121 W. Michigan St., Indianapolis 2, Ind.
Dr. C. M. Fraleigh, Univ. of West Virginia, Morgantown, W. Virginia.

COMMITTEE NO. IV

School Clinical Procedures. Curriculum Time and Sequence Necessary to Adequately Teach Periodontics Within the Confines of all Fields of Dentistry.

Consultant: Dr. R. E. Coleman

Chairman: Dr. Erwin Schaffer, 1153 Medical Arts Bldg., Minneapolis 2, Minn.
Secretary: Dr. Robt. Reeves, Univ. of So. Calif., Sc. of Dent., 925 W. 34th St., L. A. 7.

Members

Dr. Edward N. Collins, U. of Nebraska, Coll. of Dent., Lincoln 8, Neb.
Dr. David Bensinger, Washington Univ., Sc. Dent., St. Louis 10, Mo.
Dr. W. B. Leonard, Howard Univ., Coll. Dent., Washington 1, D. C.
Dr. J. W. Neilson, The Univ. of Manitoba, Winnipeg, Canada.
Dr. Robt. L. Parrish, Univ. of Tennessee, Coll. of Dent., Memphis 3, Tenn.
Dr. Jacoby Rothner, Temple U., Sc. of Dent., Philadelphia 40, Penna.
Dr. Stanley R. Suit, Western Reserve Univ., 2165 Adelbert Rd., Cleveland 8.
Dr. Henry Trowbridge, Univ. of Calif., U. C. Medical Center, S. F. 22, Calif.
Dr. F. B. Wiebusch, Medical College of Virginia, Richmond 19, Virginia
Dr. Benjamin L. Lynch, Creighton U., Sch. of Dent., Omaha 2, Nebr.

COMMITTEE NO. V

Teaching Prevention and Patient Responsibility. Consideration of Epidemiology, and Utilization of Devices to Educate the Public Through the Profession by Educating the Undergraduate.

Consultant: Dr. R. Gordon Agnew

Chairman: Dr. C. H. Williams, Univ. of Toronto, Faculty of Dent., Toronto, Canada
Secretary: Dr. Joseph Henry, Howard Univ., Coll. of Dentistry, Washington 1, D. C.

Members

Dr. Ernest Baker, Univ. of Calif., U. C. Medical Center, S. F. 22, Calif.
Dr. Clarence Butler, Coll. of Physicians & Surgeons, 344-14th St., S. F.
Dr. R. B. Cameron, Univ. of Alberta, Edmonton, Alberta, Canada.
Dr. Jack Curran, Univ. of Calif., U. C. Medical Center, S. F. 22, Calif.
Dr. Patrick Ferrillo, St. Louis Univ., Sc. Dent., St. Louis 4, Mo.
Dr. Peter D. Ferrigno, Georgetown Univ., Sc. Dent., Washington 7, D. C.
Dr. Genevieve Roth, Univ. of Kansas City, Kansas City, Mo.
Dr. Wm. Quinn, Univ. of Detroit, 630 Jefferson Ave., East, Detroit 26, Mich.
Dr. Richard Bradley, State Univ. of Iowa, Coll. of Dent., Iowa City, Iowa.

COMMITTEE NO. VI

Utilization of Research in Undergraduate and Graduate Teaching of Periodontology. A Survey of Research Projects and Needs Directed Toward Teaching and Clinical Application.

Consultant: Dr. Willard Fleming

Chairman: Dr. Irving Glickman, 483 Beacon St., Boston 15, Mass.
Secretary: Dr. Sigurd Ramfjord, Univ. of Michigan, Sc. of Dent., Ann Arbor, Mich.

Members

Dr. Frank Everett, Univ. of Oregon Dental Sc., Portland 1, Oregon.
Dr. Edward T. Fischer, Tufts Univ., 136 Harrison Ave., Boston, Mass.
Dr. Wm. Brown Ingersoll, Georgetown Univ., Washington 7, D.C.
Dr. Gerald Ivancie, State Univ. of Iowa, Coll. of Dent., Iowa City, Iowa.
Dr. Alfred Ogilvie, Univ. of Washington, Sc. of Dent., Seattle 5, Wash.
Dr. Gilbert J. Parfitt, Univ. of Alabama, Birmingham 3, Alabama.
Dr. Georges Pelletier, Univ. of Montreal, Montreal, Canada.
Dr. Douglas Pingree, Univ. of Calif., Sc. Dent., U. C. Med. Ctr., S. F. 22, Calif.
Dr. Marvin Sugarman, Emory Univ., 106 Forest Ave., N.E., Atlanta 3, Ga.
Dr. Frank M. Wentz, Chicago Coll. of Dental Surgery, 1757 W. Harrison, Chicago.

This Workshop was sponsored financially, largely by The Procter & Gamble Company with assistance from The Academy, Lactona Inc., The Fund for Dental Education and the University of California. A full report concerning some of the conclusions and activities of the Workshop will be presented to the Academy by the Chairman of the Organization Committee Dr. Harold G. Ray at the annual meeting in Dallas in October. The Organization Committee which worked hard and diligently to consumate this Workshop was composed of Dr. Maynard K. Hine, President Donald Kerr, Treasurer James Aiguier, Dr. Robert Reeves and Dr. Willard C. Fleming. The Academy was represented by its President Dr. Donald Kerr, its Secretary Dr. Clarke Chamberlain, its Treasurer Dr. James Aiguier and one of the members of the Council Dr. Evert Archer. Our Academy should take pride in this tangible evidence of the pursuit of its objective with a realization that it is the originator in the sponsorship and promotion of better teaching in its field by a representative specialty organization.

TENTATIVE PROGRAM FOR THE 1958 MEETING OF THE AMERICAN ACADEMY OF PERIODONTOLOGY IN DALLAS

Thursday, November 6

A.M.

11:00 to 11:45	Management of abnormal bleeding tendencies. Dr. J. M. Hill, Wadley Research and Blood Center, Dallas
11:45 to 12:00	Question and answer period

P.M.

2:00 to 2:30	Epidemiology and index of periodontal disease: the approach of an epidemiologist. Dr. John C. Greene, U.S.P.H.S., Washington, D. C.
2:30 to 3:00	Epidemiology and index of periodontal disease: the approach of a periodontist. Dr. C. Williams, Toronto
3:00 to 3:20	Evaluation and discussion of the viewpoints given by Dr. Greene and Dr. Williams Dr. C. Marshall-Day, Boston
3:20 to 3:45	Discussion open to floor
3:45 to 4:30	Report from the San Francisco Workshop Dr. H. C. Ray, San Francisco
4:30 to 5:00	Discussion open to floor.

Friday, November 7

A.M.

9:00 to 9:30	Biology of collagen Dr. M. Ziff, University of Texas Southwestern Medical School, Dallas.
9:30 to 9:40	Periodontal significance of collagen. Dr. P. Boyle, Western Reserve University, Cleveland
9:40 to 9:50	Open discussion
9:50 to 10:20	Biology of cementum Dr. H. A. Zander, Rochester, New York
10:20 to 10:30	Discussion of previous paper Dr. B. Orban, Colorado Springs
10:30 to 10:40	Discussion open to floor
10:40 to 11:05	Studies in periodontal healing and regeneration Dr. M. L. Morris, New York
11:05 to 11:30	Studies in periodontal healing and regeneration Dr. W. J. Linghorne, Toronto
11:30 to 12:00	Discussion open to floor Chairman for Friday morning session Dr. H. M. Goldman

P.M.

2:00 to 2:30	Newer concepts in periodontal bacteriology Dr. B. G. Bibby, Rochester, New York
2:30 to 2:40	Discussion of previous paper Dr. J. B. MacDonald, Boston
2:40 to 2:50	Discussion from floor
2:50 to 3:20	Calculus formation Dr. I. D. Mandel, New York
3:20 to 3:30	Open discussion Chairman Dr. H. A. Zander, Rochester, New York
3:30 to 5:00	Panel on: Why eliminate periodontal pockets? Moderator: Dr. B. O. A. Thomas, Palo Alto Members of the panel: Dr. H. J. Leonard, New York Dr. S. Schluger, Seattle Dr. R. H. Blanquie, San Francisco Dr. M. K. Hine, Indianapolis Each member of the panel will give his viewpoints in a 10-minute lecture. Then there will be open discussion, and the final 15 minutes will be used for a summary by Dr. B. O. A. Thomas.

Saturday, November 8

A.M.

9:00 to 11:30	Panel on: Success or failure in periodontal therapy Moderator: Dr. F. E. Beube, New York Members of the panel: Dr. I. Hirschfeld, New York Dr. H. M. Goldman, Boston Dr. S. Sorrin, New York Dr. R. Chace, Orlando Dr. M. Amsterdam, Philadelphia Each member of the panel initially will be given 15 minutes to state the basis for success or failure in periodontal therapy and to answer questions: 1. What determines whether or not teeth with periodontal disease can be saved? 2. What principles determine the indications for splinting teeth and replacing lost teeth in periodontal therapy? 3. When is palliative periodontal therapy indicated? Following these introductory lectures, there should be ample time for discussion between members of the panel and open discussion from the floor.